



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

### Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

### About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

COUNTWAY LIBRARY

HC 2EMJ 0

BOSTON  
MEDICAL LIBRARY  
8 THE FENWAY

Are these intervals see p 210



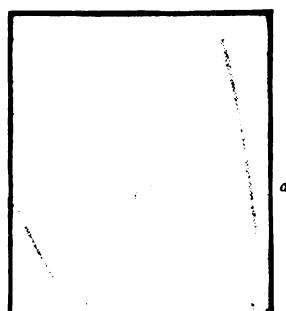


FIG. 1.



Luetin reaction. *a*, Papular form (48 hours);  
*b*, Pustular form (96 hours). *Page 89.*

FIG. 2



Egg albumen reaction. *a*, Negative—6 minutes  
after scarification; *b*, Positive—6 minutes after  
scarification. *Page 82.*

# INTERNATIONAL CLINICS

A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND  
ESPECIALLY PREPARED ORIGINAL ARTICLES  
ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIATRICS,  
OBSTETRICS, GYNÆCOLOGY, ORTHOPÆDICS,  
PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,  
OTOLOGY, RHINOLOGY, LARYNGOLOGY,  
HYGIENE, AND OTHER TOPICS OF INTEREST  
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION  
THROUGHOUT THE WORLD

EDITED BY

H. R. M. LANDIS, M.D., PHILADELPHIA, U.S.A.

WITH THE COLLABORATION OF

CHAS. H. MAYO, M.D.  
ROCHESTER

SIR WM. OSLER, BART., M.D., F.R.S. RUPERT BLUE, M.D., D.P.H.  
OXFORD WASHINGTON, D. C.

FRANK BILLINGS, M.D. JOHN G. CLARK, M.D.  
CHICAGO PHILADELPHIA

A. MCPHEDRAN, M.D. JAMES J. WALSH, M.D.  
TORONTO NEW YORK

J. W. BALLANTYNE, M.D. ARTHUR F. BEIFELD, M.D.  
EDINBURGH CHICAGO

CHARLES GREENE CUMSTON, M.D.  
GENEVA

WITH CORRESPONDENTS IN MONTREAL, LONDON, PARIS,  
AND GENEVA

---

VOLUME III. TWENTY-EIGHTH SERIES, 1918

---

PHILADELPHIA AND LONDON

J. B. LIPPINCOTT COMPANY

1918

TON MEDICAL  
LIBRARY

NOV 2 1918

TON MEDICAL  
LIBRARY

NOV 2 1918

MEC

1521  
COPYRIGHT, 1918

BY

J. B. LIPPINCOTT COMPANY

PRINTED BY J. B. LIPPINCOTT COMPANY, PHILADELPHIA, U. S. A.

## CONTRIBUTORS TO VOLUME III (TWENTY-EIGHTH SERIES)

---

**CHRISTIAN, HENRY A., M.D.**, Peter Bent Brigham Hospital.

**CUMSTON, CHARLES GREENE, B.S., M.D.**, Privat-docent at the University of Geneva; Honorary Member of the Surgical Society of Belgium; Fellow of the Royal Society of Medicine of London; Member of the Medical Society of Geneva, etc., etc., Geneva, Switzerland.

**ERDMANN, JOHN F., M.D.**, Post-Graduate Medical College and Hospital, New York.

**FISHER, LEWIS, M.D.**, Philadelphia, Pa.

**Flagg, P. J.**

**FOSTER, G. S., M.D.**, Surgeon and Pathologist to the Hospital Notre Dame de Lourdes, Manchester, N. H.

**HESS, JULIUS H., M.D.**, Department of Pediatrics, University of Illinois, College of Medicine.

**LANDIS, H. R. M., M.D.**, Henry Phipps Institute, Philadelphia, Pa.

**LEVINSON, A., M.D.**, Department of Pediatrics, University of Illinois, College of Medicine.

**LOEWENBERG, S. A.**, Philadelphia, Pa., Associate in Clinical Medicine, Jefferson Medical College; Assistant Visiting Physician, Jefferson Hospital; Visiting Physician, Philadelphia Hospital, and Eagleville Sanatorium for Consumptives.

**LYDSTON, G. FRANK, M.D.**, Formerly Professor of Genito-Urinary Surgery and Syphilology, Medical Department State University of Illinois, Chicago.

**MARGOLINE, E., M.D.**, Geneva, Switzerland.

**MARQUEZ, MAURICE, M.D.**, Montpellier, France.

**MATORINE, O., M.D.**, Moscow, Russia.

**MACKEHNIE, HUGH N., A.B., M.D., C.M.**, Loyola University Medical School; Clinical Congress of Surgeons, Chicago.

**MC PHERSON, ROSS, M.D.**, The Lying-in Hospital, New York.

**PATTERSON, ROSS V., M.D.**, Assistant Professor of Medicine, and Physician-in-Charge, Department of Electrocardiology, Jefferson Medical College, Philadelphia.

**POPE, CURRAN, M.D., Louisville, Ky.**

**REHFUSS, MARTIN H., M.D., Philadelphia, Pa.**

**REY, PAUL, M.D., Grenoble, France.**

**REYNOLDS, CECIL E., M.R.C.S., Eng.**

**WALSH, JAMES J., M.D., Ph.D., Medical Director of The Sociological Department  
of Fordham University, and Professor of Physiological Psychology at the  
Cathedral College, New York City.**

**WILLMOTH, A. D., A.M., M.D., Louisville, Ky.**

## CONTENTS OF VOLUME III (TWENTY-EIGHTH SERIES)

### CLINICS

	PAGE
CLINIC OF DR. HENRY A. CHRISTIAN, PETER BENT BRIGHAM HOSPITAL .....	1
AURICULAR FIBRILLATION. By Ross V. PATTERSON, M.D. ....	20
FRACTIONAL GASTRIC TUBE—THE TECHNIC USED IN VARIOUS GASTRIC PROCEDURES. By MARTIN H. REHFUSS, M.D. ....	40
A LECTURE ON ANEURYSMAL HÆMATOMA. By CHARLES GREENE CUMSTON, B.S., M.D. ....	55
PULMONARY TUBERCULOSIS AND CONDITIONS SIMULATING IT. By H. R. M. LANDIS, M.D. ....	66
CLINIC OF DR. JULIUS H. HESS AND A. LEVINSON .....	82
CLINIC AT THE LYING-IN HOSPITAL, NEW YORK, APRIL 30, 1918. By Ross McPHERSON, M.D. ....	100
CLINIC FOR MEDICAL OFFICERS OF THE ARMY AND NAVY, AT THE POST-GRADUATE MEDICAL COLLEGE AND HOSPITAL, NEW YORK, MAY 3 AND 6, 1918. By JOHN F. ERDMANN, M.D. ....	113
SURGICAL CLINIC OF HUGH N. MACKECHANIE, A.B., M.D., C.M., AT LOYOLA UNIVERSITY MEDICAL SCHOOL. CLINICAL CONGRESS OF SURGEONS, CHICAGO .....	137
THE NON-SURGICAL TREATMENT OF ENLARGED PROSTATE. By G. FRANK LYDSTON, M.D. ....	148

### MEDICINE

A CASE OF ACROMEGALY OF APPARENTLY ACUTE ONSET. By S. A. LOEWENBERG .....	154
A CLINICAL AND PATHOGENIC STUDY OF FAMILIAL CHOLEMIA. By PAUL REY, M.D. ....	158
SENILE EPILEPSY, WITH THE REPORT OF FOUR CASES. By O. MATORNE, M.D. ....	174

### OPHTHALMOLOGY

LATE POST-TRAUMATIC ATROPHY OF THE OPTIC NERVE. By E. MARGOLINE, M.D. ....	186
--	-----

## RHINOLOGY

SINUITIS AND HEADACHE—TREATMENT. BY LEWIS FISHER, M.D. .... 200

## SURGERY

ANÆSTHESIA IN EUROPE ON THE WESTERN BATTLE FRONT. BY  
P. J. FLAGG ..... 210

FURTHER STUDY OF THE EFFECTS OF PAIN ON THE CELLS OF THE  
CENTRAL NERVOUS SYSTEM. BY G. S. FOSTER, M.D. .... 229

THE OPERATIVE TREATMENT OF CHRONIC POLIOENCEPHALITIS  
SUPERIOR. BY CECIL E. REYNOLDS, M.R.C.S., Eng. .... 243

FRACTURES OF THE FUNDUS OF THE COTYLOID CAVITY WITH  
INTRAPELVIC DISLOCATION OF THE HEAD OF THE FEMUR. BY  
MAURICE MARQUEZ, M.D. .... 248

CHOLELITHIASIS AND CHRONIC PANCREATITIS SIMULATING  
GASTRIC CARCINOMA. BY CURRAN POPE, M.D., AND A. D. WILLMOTH,  
A.M., M.D. .... 260

WHAT SURGERY OWES TO MILITARY SURGERY: A GREAT PIONEER  
IN CLINICAL SURGERY. BY JAMES J. WALSH M.D., Ph.D. .... 274

# LIST OF ILLUSTRATIONS TO VOLUME III

## (TWENTY-EIGHTH SERIES)

### COLORED PLATES

	PAGE
Luetin reaction (Fig. 1) .....	<i>Frontispiece</i>
Egg albumen reaction (Fig. 2) .....	<i>Frontispiece</i>
Diphtheria toxin reaction (Schick test) (Fig. 3) .....	84

### PLATES AND CHARTS

	PAGE
Four-hour chart of patient P. B. B. H. Med. No. 7115 (Fig. 1) .....	4
Four-hour chart of patient P. B. B. H. Med. No. 7427 (Fig. 2) .....	12
Electrocardiographic curves (Fig. 2) .....	24
Auricular fibrillation with marked cardiac failure (Fig. 3) .....	26
Auricular fibrillation (Fig. 4) .....	27
Auricular fibrillation with normal rate (Fig. 5) .....	27
Auricular fibrillation. Mitral stenosis (Fig. 6) .....	29
Auricular fibrillation. Mitral stenosis. Advanced chronic myocardial degeneration. Sclerosis of the bundle of His. Angina (Fig. 7) .....	30
Auricular fibrillation (Fig. 8) .....	31
Auricular fibrillation (Fig. 9) .....	31
Auricular fibrillation (Fig. 10) .....	32
Normal sinus rhythm (Fig. 11) .....	32
Auricular fibrillation (Fig. 12) .....	35
Digitalis effects in auricular fibrillation (Fig. 13) .....	35
Digitalis effects in auricular fibrillation with rupture of compensation (Fig. 14) .....	36
Digitalis effects No. 2 (Fig. 15) .....	37
Digitalis effects No. 3 (Fig. 16) .....	37
Digitalis coupling in auricular fibrillation. Pseudobradyardia (Fig. 17) .....	38
Auricular fibrillation. Mitral stenosis. Marked cardiac failure (Fig. 18) .....	38
Auricular fibrillation. Bradycardia. Extreme strophanthus effects (Fig. 19) .....	39
Tube in position (Fig. 1) .....	41
Method of aspiration (Fig. 1 A) .....	42
Digestive and indigestive periods (Fig. 2) .....	44
Tips used with tube (Fig. 3) .....	46
Murphy drip (Fig. 4) .....	49
Syphon method of obtaining gastric secretion (Fig. 5) .....	51
Double gastro-duodenal tube in position (Fig. 6) .....	52
Possibilities in the evolution of the secretory curve of digestion (Fig. 7) .....	53
Congenital syphilis (Fig. 4) .....	90

Michael E. Philadelphia Hospital. Acromegaly, showing massive features (Fig. 1) .....	156
Michael E. Philadelphia Hospital. Acromegaly, showing massive nose, protrusion of lower jaw, and prominent orbits (Fig. 2) .....	156
Michael E. Philadelphia Hospital. Acromegaly, showing curvature of spine and shortness of neck (Fig. 3) .....	156
Case of emphysematous chest (Fig. 4) .....	156
Showing characteristic enlargement of fingers (with round nails), toes and tibia (Fig. 5) .....	156
Case of pulmonary hypertrophic osteoarthropathy (Fig. 6) .....	156
Acromegaly, showing periosteal thickening of phalanges and enlargement of soft parts (Fig. 7) .....	156
Michael E. Philadelphia Hospital. Acromegaly, showing periosteal thickening along metatarsals and increase in soft parts (Fig. 8) .....	156
Michael E. Philadelphia Hospital. Acromegaly, showing posterior clinoid hypertrophied, anterior clinoid enlarged (Figs. 9 and 10) .....	156
Guinea pig operated upon under ether anaesthesia only. Section of cord. Great number of nephelated cells (Fig. 1) .....	230
Guinea pig operated upon under ether anaesthesia only. Section of cord. Clearly defined nephelation and vacuolation (Fig. 2) .....	230
Guinea pig operated upon under ether anaesthesia only. Section of cord. Great number of well-defined nephelated cells (Fig. 3) .....	230
Guinea pig operated upon under complete narcosis. Section of cerebral cortex (Fig. 4) .....	230
Guinea pig operated upon under complete narcosis and amnesia. Section of cerebral cortex (Fig. 5) .....	230
Guinea pig operated upon under complete narcosis and amnesia. Section of cerebral cortex (Fig. 6) .....	230
Guinea pig operated upon under local analgesia alone. Section of cord. (Fig. 7) .....	230
Guinea pig operated upon under local analgesia alone. Section of cord (Fig. 8) .....	230
Guinea pig operated upon under local analgesia alone, producing complete nerve block. Section of cerebral cortex (Fig. 9) .....	230
Showing production of mother circle of analgesia with progressively formed daughter arcs (Fig. 10) .....	232
Guinea pig killed with lethal dose of morphine (Fig. 11) .....	236
Guinea pig is given no narcotic, anaesthesia or other supporting measures (Fig. 12) .....	236
Guinea pig under ether anaesthesia, (Fig. 13) .....	236
Guinea pig under chloroform anaesthesia (Fig. 14) .....	236
Guinea pig under ethyl-chloride anaesthesia (Fig. 15) .....	236
Guinea pig under nitrous-oxide anaesthesia (Fig. 16) .....	236
Guinea pig under nitrous-oxide-oxygen anaesthesia (Fig. 17) .....	236
Guinea pig under nitrous-oxide-oxygen-ether anaesthesia (Fig. 18) .....	238
Guinea pig. Novocaine infiltration, nerve block (Fig. 19) .....	238

## LIST OF ILLUSTRATIONS TO VOLUME III

ix

Guinea pig. Stovaine in spinal canal (Fig. 20).....	238
Cobweb construction to show direct ratio between pain nerve transmission and the anesthesia used (Fig. 21).....	239
First, second, third and fourth degrees of pain nerve transmission; first end control; further end control (Fig. 22).....	240
Case of polioencephalitis superior in a fit (Figs. 1 and 2).....	244
Former hemiplegic imbecile three months after operation (Fig. 3) .....	245
Piece of dura mater removed several months after operation (Fig. 4).....	246
Pial aspect of dura shown in Fig. 4 (Fig. 5).....	247
Line of junction between new and old dura as shown by transmitted light (Fig. 6) .....	247
Fracture of the fundus of the left cotyloid cavity without fracture of the femoral neck (Fig. 1).....	249



WAR INDUSTRIES BOARD  
WASHINGTON

W. M. BARUCH  
CHAIRMAN

IN YOUR REPLY  
REFER TO \_\_\_\_\_

From: Lieut. Colonel F. F. Simpson, M.C., N.A.,  
Chief of Section of Medical Industry.

To: The Doctors and Dentists of the Country.

Subject: Utilization of Platinum in Unused Instruments.

1. In view of the limited supply of platinum in the country and of the urgent demand for war purposes, it is requested that every doctor and dentist in the country go carefully over his instruments and pick out EVERY SCRAP OF PLATINUM that is not absolutely essential to his work. These scraps, however small and in whatever condition, should reach Governmental sources without delay, through one of two channels:

- (a) They can be given to proper accredited representatives of the Red Cross who will shortly make a canvas for that purpose.
- (b) They may be sold to the Government through any bank under the supervision of the Federal Reserve Board. Such banks will receive and pay current prices for platinum.

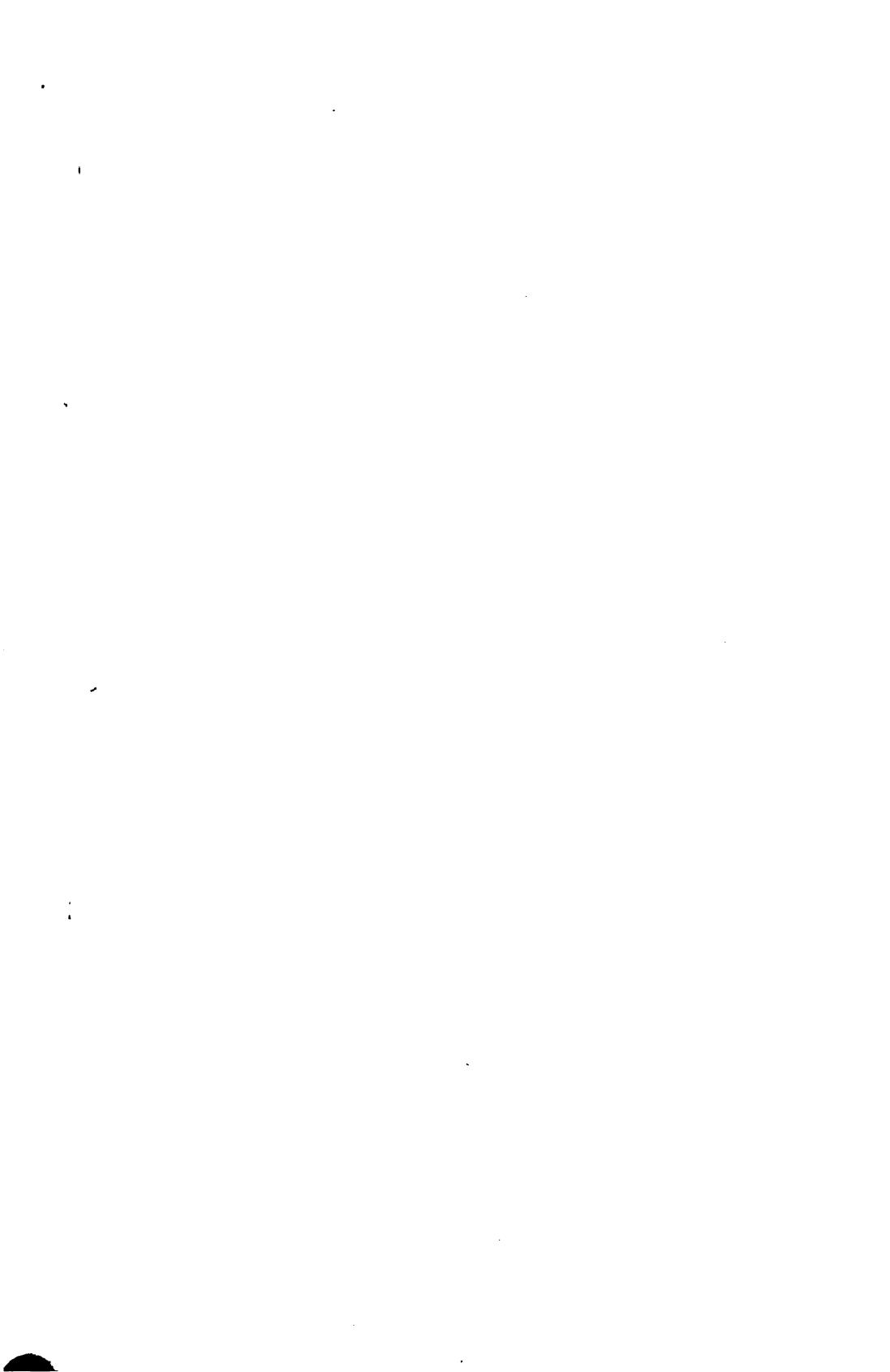
By giving this immediate attention you will definitely aid in the war program.

2. It is recognized that certain dental and surgical instruments requiring platinum are necessary, and from time to time platinum is released for that purpose. It is hoped, however, that every physician and every dentist will use substitutes for platinum for such purposes wherever possible.

3. YOU ARE WARNED against giving your scrap platinum to anyone who calls at your office without full assurance that that individual is authorized to represent the Red Cross in the matter.



Lieut. Col. F. F. Simpson, M.C., N.A.,  
Chief of Section of Medical Industry.



STON MEDICAL

NOV 2 1918

LIBRARY  
Clinics

CLINIC OF DR. HENRY A. CHRISTIAN  
PETER BENT BRIGHAM HOSPITAL

ACUTE VEGETATIVE ENDOCARDITIS

DR. CHRISTIAN to students: I wish you would take the patient in the room under the seats (P. B. B. H. Med., No. 7427) and go over her.

Dr. Christian to another student: I want you to take this patient's pulse (P. B. B. H. Med. No. 7115) first, and when you have finished listen to his heart.

Dr. Christian: The patient here is twenty-eight years of age (P. B. B. H. Med. No. 7115). He was admitted to the hospital on August 20. He has been here continuously since then. He came in complaining of weakness, anorexia, high fever and drenching sweats. He was born in Russia and has lived in the United States for five years. His family history is negative. He has been married for two years. His wife is healthy. He has one child who is healthy. His habits are good. Up to January of this year he worked as a clerk in a small store. The work was not hard nor tiring. Since January of this year he has had a somewhat harder job in a wholesale hardware store. Three years ago he was at the Boston City Hospital with what he thinks was typhoid and pneumonia. He was there for eight weeks. After going home it was four weeks before he could go back to work. He has had frequent sore throats. He has never had rheumatism, scarlet fever, pleurisy or malaria. There has been no known association with tuberculosis. There is no history of any acute infections of any kind other than those I have already given. His past history is negative—he has been a healthy man. He weighed 210 pounds before the present illness began. He has lost weight until his weight was down to 148 pounds just before he came

in. This was a gradual loss of weight over a period of six months. During all of that time he was only a little bit sick. During the last two months before coming in his loss of weight was a little more rapid.

During that time he was more definitely ill; that is, he was having fever, sweats, etc. For three years he has had a cough yielding a small amount of white, clear sputum. He has never been sick on account of this cough. He was never examined by a physician. Six months before he came in (that was about January 1, when he changed his job) he began to have more cough and raised a large amount of thin, watery sputum. He did not go to see a physician at that time, or at least if he did he did not have a physical examination. About June 1, of this year, he rather suddenly developed a high fever in the afternoon which continued and was frequently as high as 104°. At that time he began to get more definitely weak and to have night sweats. His temperature, which was anywhere from 102° to 104° in the afternoon, was more nearly normal in the morning, apparently some mornings it was normal. He was seen then by a physician and was sent to the Jewish Hospital to have a thorough examination. From there he was sent to the Boston Dispensary for an X-ray of his chest. According to him the picture showed definite evidence of pulmonary tuberculosis. He was sent from the Jewish Hospital to Rutland (a State tuberculosis sanatorium). He went home, however, and remained at home for three weeks before he went to Rutland, possibly having some difficulty in getting in. During the three weeks he was at home his temperature was taken regularly and he always has this very high evening fever. At Rutland they were unable to find any pulmonary lesion, and after remaining there for some time he was advised to come back to Boston and go to some hospital for a general examination. Apparently they did not feel that he had pulmonary tuberculosis. He followed their advice as far as going home was concerned and also followed their advice in regard to the hospital and came in here and was admitted on August 20, 1917, and has been here since. Since he has been here he has continued to do apparently what he began to do on June 1; namely, run a fever which sometimes is a continuous fever without much fluctuation and at other times is a more definitely fluctuating temperature. There has been very little change in that

since he has been here. (Demonstrations of charts.) You see sometimes these up and down excursions are very great and at other times the temperature is very uniformly up. That is shown better in the four-hour chart (Fig. 1), a sheet of which I will pass around. The temperature at times has been as high as 105° and 105.5° in the afternoon. In the morning it is frequently normal; it is occasionally subnormal. So there is a fever of known long duration. Apparently it was pretty accurately taken before he came to the hospital from June 1 to August 20, and there has been a continuation of the same from August 20 to date, November 5.

Dr. Christian to students: What do you make out in regard to the pulse and to his heart?

Student: The pulse is normal but the artery does not come up well.

Dr. Christian: It is a small pulse but there is nothing abnormal about the pulse wave. It is a soft pulse and probably a little rapid, but not a particularly rapid pulse. How about his heart?

Student: The heart sounds are a little muffled. I did not hear any murmurs.

Dr. Christian: The heart sounds are a little muffled, but there are no murmurs to be heard. How about the size?

Student: It goes away out into the axilla.

Dr. Christian: By percussion or palpation?

Student: By palpation.

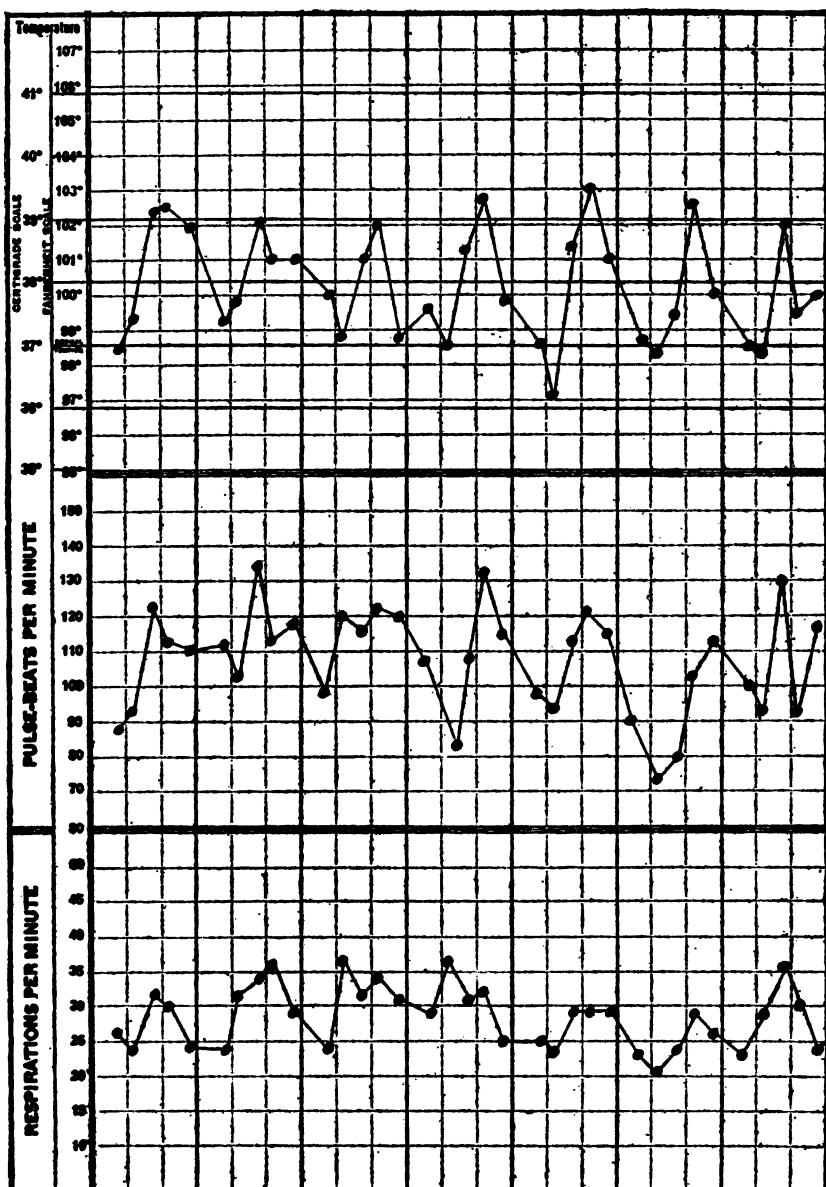
Dr. Christian: That is correct. As far as auscultation is concerned the sounds are muffled, weak or distant—there are various ways of expressing it. The first and second sounds are similar in quality and there is no murmur to be made out. In regard to the size of the heart you feel a pulsation out to about the anterior axillary line, rather high as I feel it here in the interspace just on the level with the nipple.

Dr. Christian: What do you make out at that point? (Left lower border of sternum.)

Student: I did not percuss it.

Dr. Christian: As we percuss we do not get the usual cardiac dulness—flatness in this area—we get resonance. He is lying somewhat tipped on the left side and he generally lies over on that side.

FIG. 1.



Four-hour chart of patient P. B. B. H. Med. No. 7115.

Dr. Christian to patient: Can you lie flat in bed?

Dr. Christian: He has a bad knee and that interferes with his motions. It may be that his heart is an easily movable one and when he is lying over on his side it tips over and that is suggested because the impulse is high, whereas ordinarily with an enlarged heart you find the impulse lower down. To put it another way, a normal-sized heart as it goes over is rotated somewhat on a fixed point here at the base, and although normal sized the apex comes up as it is tipped over.

Dr. Christian to student: See if you can locate the impulse.

Student: It is always well to take into consideration the position of the patient, whether lying on one side or the other, in locating the apex or the border on percussion because the heart in a perfectly normal individual moves a good deal. In percussing with the patient first on one side and then on the other side you find the apex normally shifts about that (three to four cm.) far.

Student: I cannot locate the apex impulse very satisfactorily.

Dr. Christian: It is pretty hard to tell where it is. It is a weak diffuse one. What about the one out here—is that gone?

Student: Yes, the impulse felt when the patient was not flat in bed has disappeared.

Dr. Christian: Now listen and see where you get the sounds best. Sometimes when it is difficult to locate the heart by percussion as to borders or by palpation you can check up your impressions by listening and getting the point at which the sounds are heard best, more distinctly or louder, remembering that sometimes consolidated lungs or fluids transmit sounds so that you hear heart sounds very distinctly outside the cardiac area, so that in an area of dulness you cannot localize always the position of the heart by the sounds.

Student: About here.

Dr. Christian: It is about in the normal position when you listen to the sounds.

Student: There is a low murmur.

Dr. Christian: Did you say you heard a murmur?

Student: I thought I heard one on expiration, but I do not know.

Dr. Christian: As we percuss that heart with the patient flat on his back the border of dulness is, quite definitely, just in the nipple

line. In other words, really his heart was shifted over somewhat but was not enlarged essentially. It got into normal position with him flat on his back. There is still resonance instead of absolute dulness over the precordial area but the dulness is greater than it was before, so the heart probably is not enlarged or if enlarged it is only very slightly enlarged. You can hear a soft, slightly blowing, systolic murmur just over the apex, but it is very slight. I won't bother you to go over his lungs. I will speak of the few findings we have had there. There is a slight occasional râle and a little dulness at the bases.

The very considerable loss of weight is evidenced in his forearm. You remember he said he weighed 210 pounds last January, and that is not the arm of a man that weighs 210 pounds by a good deal now. When he came in his physical signs were practically what they are to-day. He showed loss of weight and he showed a heart that was normal in size. The day he came in a soft, systolic, blowing murmur was heard over the precordium. His blood-pressure was  $\frac{150}{85}$ . He had a haemoglobin of 74 per cent., red cell count of 4,256,000 and a leucocytosis of 14,400. Those have been his physical signs in very large part ever since he came in. On the next day, August 21, examination of the heart was negative; there were no murmurs; abdomen was negative; spleen was not felt; liver edge was easily felt on inspiration. On the 23d the note was made, "Pulmonary signs negative except for occasional fine crackles throughout the right chest front and back. These râles, however, are not constant. Sounds of the heart are rapid and of good quality. Systolic murmur heard over the precordium, of greatest intensity at the base. Liver edge felt from the fourth interspace to second fingers below the costal margin in the right nipple line. Spleen not palpable." On August 29 this note was made, "Sounds rapid, regular, tic-tac quality. No murmurs. Percussion of both lungs shows slight relative dulness over the lower portion, front and back, and on deep respiration a few fine crackles are heard. Respiration on the whole shallow and not rapid. Patient not cyanotic. No areas of bronchial breathing. Respiration in general rather faint and distant." The notes read, as far as physical examination is concerned, just like those made by various members of the staff, who have gone over him since he came in on August 20.

Two X-rays of his chest are negative. One was taken on August

21, and the other one was taken on September 14. We had a third plate, taken quite recently, which showed that the heart is not enlarged. The diaphragm is rather high and possibly the heart border is a trifle out to the left, but there is no very definite enlargement, and there is no change in the heart size in the different plates. There is the normal amount of thickening along the bronchial tree, but the apical portion of both lungs and the peripheral portion of both lungs are entirely clear, and there is no evidence of infiltration of any kind. The third plate was not one in which the patient was lying squarely on his back. It showed a definite deformity due to the fact that the sternum had rotated to one side and the vertebrae to the other side, and near the sternal margin there was a round area which was pretty dense and a little bit larger than the end of my thumb, and the rest of the plate was entirely negative. The question comes as to whether that round area of increased density was more than what we ordinarily see in plates because in this region there is the regular density of the hilum of the lung and frequently enlarged peribronchial lymph nodes, and it may be that that particular point in the rotation was brought out in the view. I am a little inclined to think that that interpretation should be made and that all three plates bringing it down to a pretty recent date are negative.

As to special examinations—an ophthalmoscopic examination made several times is negative. During his stay in the hospital he has been running a varying leucocyte count: 14,400 when he came in, one count which was as high as 38,700 on October 7, and two or three days later than that, on October 10, a count of 10,800. Those represent his extreme variations. Blood cultures were taken on August 21, September 8, September 13, and on October 19, and all of those blood cultures were negative. His sputum has been repeatedly examined recently. In early tests there was very little sputum. These examinations have been negative for acid fast organisms. There is nothing particular about these sputum examinations. Recently he has begun to cough up bloody sputum. This is some sputum that he coughed up this morning. You see it is mostly blood, but in it are grayish masses of pus. That specimen has not been examined yet for tubercle bacilli. That development of bloody sputum is a quite recent affair. (Subsequent examination showed no tubercle bacilli.)

The only other thing that has happened during his stay in the hospital is the development of some fluid in his abdomen in the latter part of August, and in the first part of September. The fluid accumulated enough so that he had shifting dulness, typical signs of fluid, and on September 13 we tapped his abdomen and got out 2750 Cc. of straw-colored fluid, with a cell count of 2400; 99 per cent. lymphocytes and 1 per cent. polymorphonuclears. Smears made from the clot of that specimen showed no tubercle bacilli, and a guinea pig injected was negative for tubercle bacilli. Just prior to that he began to develop some signs suggesting a meningeal irritation, a positive Kernig sign, some headache, etc., so that on September 10 lumbar puncture was done and a normal amount of fluid was obtained, having a normal cell count and a slightly positive globulin test. Since his abdomen was tapped the fluid has not reaccumulated. Subsequently he has had no more signs of meningeal irritation. The Wassermann reaction in blood and spinal fluid is negative. The Von Pirquet skin reaction is negative.

You notice that he has a bandage on his forearm just above the elbow and I spoke of his having some trouble in his knee. Those represent subcutaneous abscesses which have been drained by incision, and the one on his knee, which was opened about October 25 or 26, showed an organism which grows as very small colonies. They grow in the bouillon as a flocculent precipitate, and if you shake the tube you see little masses of bacteria that float. Culturally and morphologically the findings justify the diagnosis of streptococcus viridans. Those organisms were obtained from the deep abscesses in the region of his knee, remembering that his blood cultures have been negative.

The only other change that has taken place in him is that the liver has quite definitely enlarged. It was made out on the first examination in the hospital as enlarged, the edge being felt not very much below the costal margin. Since then it has increased in size and this morning was noted to be tender. It is not a very large liver but it is one that extends pretty well down into his abdomen and recently has been tender. The spleen has never, I think, been felt. The other day in feeling his abdomen I felt something to the left of the median line which I thought might have been spleen but followed across, it seemed more like an enlarged left lobe of the liver than the

spleen, so it is fair to say that as far as physical examination goes the spleen has never been felt.

The patient's essential feature has been the fever. The fever has existed since June 1 to date and it has been an up and down fever. His physical signs have been essentially negative—there is no definite evidence of a cardiac lesion, no definite evidence of a pulmonary lesion, occasionally a few râles are heard in the lung, but they have been inconstant. After the patient has been lying flat on his back there is some slight dulness at the base of the lung, and râles there. X-ray examination showed no pulmonary lesion.

The question comes up as to what is the probable diagnosis in a patient with that type of temperature and so few physical signs. It practically always lies between three things: syphilis, miliary tuberculosis and vegetative endocarditis. Syphilis is the least frequent cause of that type of temperature and there is nothing in this patient, either Wassermann reaction or history, to suggest syphilis. Probably miliary tuberculosis is the more common cause of that type of temperature. In this patient we have a negative guinea pig test on his abdominal fluid as far as tuberculosis is concerned, a negative Von Pirquet reaction, which when negative in the presence of fever is not worth very much, and it happens to be negative here, and a spinal fluid which was normal, showing no evidence at that time of tuberculosis of the meninges, and an ophthalmoscopic examination which showed no miliary tubercles.

A short time ago it was suggested that it might be of interest to have a complement fixation test done for the reaction with tubercle bacilli as antigen and we sent his serum up to Saranac Lake, where they are doing a good deal of work on that reaction, and got back the report that they would be very much interested to know what would turn out eventually in the case, but the serum was negative for tuberculosis. The other thing is that miliary tuberculosis usually, if it is producing the temperature, produces very definite physical signs in the lungs or signs of meningeal tuberculosis within a shorter period of time than the interval between June 1, when his symptoms began, and November 5 (to-day), and none of those signs have appeared. When he got the stiff neck, etc., we thought he was beginning to have meningitis, but no progress has been made on that score.

It is of interest to go back and see what diagnoses were suggested as the patient went along. On August 20 a house officer, after taking the history and making a physical examination, thought it was miliary tuberculosis. On August 21 one visiting physician said: "My diagnosis would be diffuse tuberculosis, probably pulmonary in origin and miliary now." On August 29, when I saw him, my interpretation was: "The temperature curve and its duration even without any cardiac signs except enlargement strongly suggests acute endocarditis as the most probable diagnosis." There are some other notes here in which no definite diagnosis is made. Another house officer said on September 18: "No diagnosis possible at this time." A little later this same house officer thought: "Recent fluid in the abdomen and general doughy feel of the abdomen rather suggests tuberculous peritonitis." On October 23 he said: "All signs taken together seem to point to a generalized tuberculous infection." On the 24th after some râles were found he added: "These findings seem to strengthen the diagnosis made on the patient of tuberculosis." November 1 I made a note: "The case has continued with fluctuating fever, no development of physical or pulmonary signs by physical examination or X-ray plate. The diagnosis of vegetative endocarditis seems most probable and is in accord with the findings of streptococcus viridans in a deep abscess about the knee joint."

The next patient (P. B. B. H. Med No. 7427) is a young woman, age twenty-four, who comes in complaining of heart trouble. Her family history is negative. Her habits are good. Her work has been light. She has been a milliner and has had rather long hours and not very good conditions surrounding the work, but for six months she has not been at that work because the one who employed her went out of business. She was born in Massachusetts and has lived here always. She had measles at six; tonsillitis at nine; chicken pox at eleven; mumps at eighteen. She has had mild attacks of pleurisy in the left upper chest, the attacks occasionally lasting two or three days, during recent years. She has had no other acute infectious diseases. Her tonsils were removed at the age of nine. She cut her left hand on a piece of glass at the age of fourteen, requiring suture, and the wounded finger has been stiff since then. A year ago she weighed 104 pounds; two weeks ago she weighed eighty-four pounds. This loss of weight has

been gradual. She was quite well up to two months ago, when she gradually began to complain of weakness. She has slept poorly at night. She has felt so weak that she has had to lie down most of the day. She knows of nothing that was the cause of this weakness. That is, the onset was gradual and insidious and she cannot attach any definite event or cause to the beginning. She had not over-worked and had not caught cold. After this weakness began she noticed a considerable amount of palpitation of the heart at night and with the palpitation she often had night sweats. These have been getting less marked recently. One month ago she went with a girl friend to the Boston Dispensary and was told at that time that she ought to go to a hospital and was told to go home and go to bed. She did so and was in bed for about two weeks. She did not feel much better as a result of that, so she came to the hospital and was admitted on October 19, 1917. She has had a fever since she has been in the hospital, in some observations it being a little above 102° in the afternoon and in the morning a little above 99°, that is, since she has been here her temperature has not been at any time normal, but it has not been elevated more than about 3° above the normal. You see from the four-hour chart (Fig. 2), like the other patient, it has been a fluctuating temperature, but there have been no such steep rises and falls as in the preceding case.

Dr. Christian to student: What did you make out?

Student: The pulse is regular and very rapid. It is full and easily palpable. The arteries are normal. There is a distinct venous pulse in the neck. The heart is not enlarged to the right, being about 3cm. from the mid-line to the right; to the left I could not percuss it—dulness goes clear over to the side of the chest. At the apex there is a pre-systolic thrill.

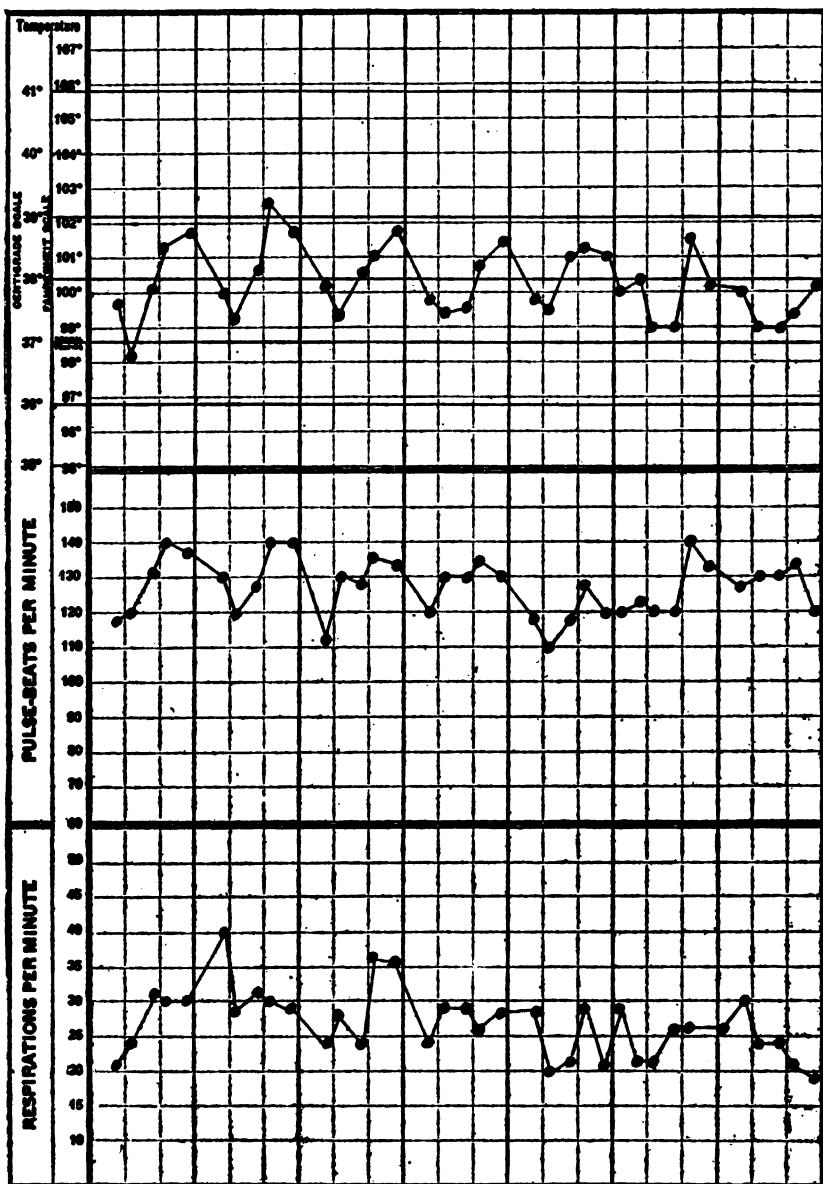
Dr. Christian: There is a systolic murmur heard over the whole precordium, maximum at the apex, and a pre-systolic murmur heard at the apex. There are some râles in the left chest. What did you hear at the left sternal border?

Student: Nothing more than I said.

Dr. Christian: Listen right over the lower left border. What do you get there?

Student: I hear a systolic murmur.

FIG. 2.



**Four-hour chart of patient P. B. B. H. Med. No. 7427.**

Dr. Christian: Is it systolic? No, there are two murmurs—the loudest and longest is diastolic. The shorter one is systolic. The patient has quite marked murmurs. There are murmurs at the apex, murmurs at the base, and a thrill. There is a systolic murmur that you can hear both at the base and at the apex, and from the middle of the sternum at about the level of the third interspace down along the left sternal border there is a loud, blowing diastolic murmur and a quite evident, shorter systolic murmur of about the same intensity, so that the patient has quite definite signs of a valvular lesion producing changes. In addition she has a definite anæmia of the secondary type; a haemoglobin of 65 per cent. and a red count of 3,400,000 when she came in on October 20, and on the last day of October the haemoglobin had dropped from 65 per cent. to 51 per cent. and the red count had dropped from 3,400,000 to 2,128,000. In addition she has a polymorphonuclear increase, giving a leucocytosis the highest 17,400 and the lowest 9200; another count 14,200.

When she came in her heart action was made out to be regular, rapid, heart enlarged, loud systolic and diastolic murmurs heard over the whole precordium; in the third interspace one inch to the left of the sternum loud and musical in quality. These murmurs are heard in the aortic area and along the left border of the sternum, with less intensity at the apex. On October 20 I made a note: "In the aortic area and over the sternum and along the left sternal border to the tricuspid area is heard a double murmur which in places becomes high pitched and musical in quality. The same murmur can be heard with less intensity at the apex. The first sound has a ringing valvular quality." On October 25 one of the visiting physicians made out the murmurs as follows: "At the apex can be heard a definite pre-systolic roll which is transmitted somewhat toward the sternum. Just inside the apex can be heard a short, soft, systolic murmur and a similar diastolic murmur. The diastolic murmur becomes much more prominent toward the base and is very loud in the third left interspace close to the sternum. The systolic murmur can be heard in this region, but is considerably fainter than the diastolic. The pulse is Corrigan in type. No capillary pulsation can be made out."

That patient has a weakly positive Wassermann reaction in her blood. A blood culture was taken on October 26. The plate showed

about twenty-five colonies—small colonies—and here are transplants from one of those colonies and on the blood serum surface you will notice very minute dry colonies, that appear just as little points, slightly glistening and scattered over the surface. You notice in the bottom of the bouillon tube a sediment which if you shake it gives some flocculent masses of precipitate, rather fine masses in suspension. That organism is characteristic of streptococcus viridans and as far as we know is streptococcus viridans.

These two cases present certain features in common and certain distinguishing characteristics. In the first place they both had an insidious, gradual onset, as far as we can make out from the histories of the patients, not marked by any definite cause and not a sudden onset. Both patients have lost weight. Both patients have had night sweats. Both patients have had fever of an irregular type, high in the afternoon. There is nothing very definite in the past history of either individual to point to a beginning of an infection. One has been surprisingly well. The other patient, the woman, has had scattered along during her childhood and young girlhood the ordinary children's diseases. She had tonsillitis at nine, and her tonsils were taken out at nine. One patient shows essentially a normal heart and the other physical signs are normal, with the exception of an enlarged liver and an occasional râle in the lung. The other patient shows marked signs of a valvular lesion of the heart, pointing to aortic insufficiency and mitral insufficiency and possibly a mitral stenosis. During the last few days the physical signs of mitral stenosis have become a little bit more evident. The important thing in that case is the diastolic murmur, pointing to either aortic insufficiency or mitral stenosis or both, and that is what a diastolic murmur means in a patient and is always important. A systolic murmur in most cases means regurgitation from the mitral valve and is ordinarily in itself of very little importance. Both patients show leucocytosis, one shows very little anaemia and the other one shows striking anaemia which has increased since the patient has been in the hospital. The first patient has had repeated negative blood cultures, but streptococcus viridans was isolated from pus in a deep subcutaneous abscess in the region of the knee. The other patient, the young woman, has a positive blood culture of streptococcus viridans. Recently the man has added to his

picture increased sputum with a considerable amount of blood, making a true haemoptysis. The condition in each is probably primarily due to the same thing; namely, vegetative endocarditis, the vegetations being due to streptococcus viridans. In the one case the vegetations apparently have been very minute and have caused no ulcerative or destructive process in the valves and up to the present have not interfered with the function of the muscle. In the other case, because this patient apparently prior to the onset of this illness had nothing pointing to a valvular lesion in the heart (I say "apparently" in the second case), the organisms have caused destruction of the valve, as far as the aortic valve is concerned, with aortic insufficiency; possibly similarly a destructive process of the mitral valve, with mitral insufficiency; possibly some reparative process going on in the mitral valve; possibly large masses of vegetations, such as we have seen recently in an autopsy, have interfered with the function of the mitral valve in the sense of producing an obstruction and giving us for one reason or the other slight signs of mitral stenosis.

The woman has had no peripheral evidences of bacterial infection, nor has she had any signs of emboli from the valves getting out into the peripheral circulation except a few petechiae in the conjunctivæ. She has had no petechiae on the skin, no abscesses, no embolic circulatory symptoms, etc. The man for a long time presented the same picture and had no signs of emboli getting away from his heart until the infection developed first around his elbow and later around his knee, which can be explained as bacterial in origin, possibly just a small amount of bacteria getting out into the peripheral circulation, lodging at that point and producing a suppuration.

In regard to the haemoptysis, he has had no extensive cyanosis, no dyspnoea, no extensive pulmonary symptoms or signs, and it is possible that the haemoptysis has taken place owing to the organism from his heart getting into his lungs and producing some sort of destructive process. There is still the possibility that he has a tuberculous lesion and that the tuberculous lesion is causing the usual ulcerative type of process and that he has haemoptysis from that cause. It is not very probable that that is the kind of haemoptysis that we have spoken of in cases of mitral stenosis or chronic myocarditis, examples of which have been shown to you, where the haemoptysis is due to chronic passive

congestion or infarction, a process that takes place when an embolus lodges in the lung in chronic passive congestion. This may be infarction in the sense of an infected embolus causing interference with the circulation to a part of the lung with some breaking down, but it is not the ordinary form of infarction with chronic passive congestion.

However, I think both cases are of the same general nature and the man particularly well illustrates the difficulty in making a diagnosis in one of these cases and the long time that the process can continue without producing any demonstrable cardiac lesion, though the primary cause is acute endocarditis. That has been pointed out by various observers and the first thing to-morrow afternoon I will run over briefly some of the points on the general subject of these long-continued cases of vegetative endocarditis. They are important and a very interesting group, particularly as to mistaken diagnoses, and a very common happening is for them to be called tuberculosis and the patient be sent to a tuberculosis hospital, as occurred in this man.

November 6, Dr. Christian: The first thing this afternoon I want to finish up a little bit more in detail the discussion of the cases that I showed you yesterday. That type of case that I showed has been variously named. Sometimes it has been spoken of as a case of "chronic infectious endocarditis." That was the term under which Dr. Billings reported a group of them in the *Archives of Internal Medicine*, in 1909, vol. iv, page 409; and, by the way, that is a very good discussion of the subject to read. There is a great deal of interest in it. The discussion itself is very brief, taking only four or five pages, but there are fourteen cases of the condition reported with not extensive notes but pretty full notes on the essential features of the condition. Then other terms have been used, such as "sub-acute bacterial endocarditis." That is the term that Dr. Libman, in New York, has used. There are two papers, one in the 1912 and the other in the 1913 volume of the *Transactions of the Association of American Physicians*, by Dr. Libman on the same subject, with a considerable number of case reports. Dr. Libman is particularly interested in the group during the bacterial free stage and Dr. Billings speaks more of them when bacteria are found in the circulation. Then in the *Quarterly Journal of Medicine*, 1909, vol. ii, page 299, Sir William Osler gives a very good description of the group. Then you will find

in these various papers a discussion of the organism that causes the condition—the organism which I spoke of as being streptococcus viridans. You will find that Billings in his paper speaks of the organisms as a type of pneumococcus. The organisms were studied at that time by Dr. E. C. Rosenow and he was inclined to regard them as a pneumococcus organism. Libman in his various papers does not give it any special name or discussion as to just what it is. They have been described occasionally as the coccus of rheumatism. There has been a great deal of discussion as to the exact classification of this organism. They are cocci that grow in chains, and present certain cultural characteristics which we ordinarily regard now as indicating one of several varieties of streptococcus. So do not feel disturbed in reading different papers to find different names given to the organism. Another term that is used very often for the diseased condition is "chronic ulcerative" or "chronic infectious endocarditis." This term really describes the condition and its varieties. Sometimes they are chronic; sometimes they are fairly acute. Sometimes there is a destructive process in the valves with signs of valve lesions, such as shown by one of our cases, and there are cases in which the vegetations are small and there are no destructive lesions, as illustrated by the other case.

A very interesting thing about the cases is the mistakes in diagnosis—the different views in regard to a diagnosis before the correct diagnosis is made. That was illustrated to a certain extent by our own case, which was sometimes regarded as a case of tuberculosis—miliary tuberculosis—and at other times as a case of endocarditis. Not infrequently the patients are regarded as cases of typhoid fever as another possibility. Not infrequently, on account of the fluctuating temperature, they are thought to be malaria. Those various diagnoses are made.

Another striking thing about the cases is that very often the condition exists for a long time with the patient in really a pretty good condition; that is, notwithstanding the fact that there is bacteræmia, fever and an active valve process going on, compensation is perfectly good, the patient feels pretty well. For instance one of Dr. Billings' cases during the febrile period went on a vacation and did some duck shooting and fishing, and many of the cases are able to travel, some

going off on long trips, etc., so that the cases may exist with relatively few symptoms. Most of the cases are eventually fatal. Even those cases that become bacteria-free and apparently remain free from bacteria for several years eventually die. I think of Dr. Libman's cases, in this second report, of the twenty-one cases which he had observed, seventeen had died during the period of observation and the diagnosis was confirmed in sixteen of those seventeen by autopsy findings.

The condition is somewhat different from what is ordinarily spoken of as malignant endocarditis, or malignant vegetative endocarditis, where the process is much more rapid, the destruction of the valves is much more extensive and the patients live a shorter period of time. The second case which I showed you is more of that type.

If you take more frequent blood cultures I suppose it is much more certain that you would get positive results but fairly frequent blood cultures may give negative results in these cases. Most of the patients have a leucocytosis, but a good many of them have a normal white cell count, so the presence or absence of a leucocytosis is not characteristic of the condition; sometimes it is present, sometimes it is absent. The most difficult cases to diagnose are those that present no cardiac symptoms and no cardiac signs.

Now, of course, the patients that I have shown you as illustrations of that may turn out to have something else and that not be a correct diagnosis of this type of vegetative endocarditis. If that is the result I will let you know later, but I have seen cases with just as little physical signs and just as few symptoms referable to the heart who eventually died, and autopsy showed vegetative endocarditis with small vegetation on the valves, and the patients did not die for a very considerable period of time after they began to run a temperature, and it was only in the end stages of the condition that they began to have septic emboli thrown off and a septic condition develop in various parts of the body, by which the condition was recognized as a vegetative type of endocarditis; and in that connection are particularly interesting the cases that Dr. Libman reports from the Mt. Sinai Hospital, in New York, where the patients did not die of cardiac disease. Some of them died a nephritic death. There is a type of glomerular lesion associated with this particular organism in this particular group of cases. The

organism in this group apparently produces a type of glomerular lesion, one described by Aschoff, in Germany, and since by Baehr, who described it while abroad, but he is now associated with Dr. Libman at the Mt. Sinai Hospital. Some of those cases present themselves with the picture of an enlarged spleen and secondary anæmia. They are called cases of splenic anæmia and the true condition is missed. Some of those cases progress downward along the lines of increasing anæmia and die the death of an anæmic, and then, of course, you get others who have chronic valvular lesions associated with or developing as the result of the vegetative process and they die the ordinary cardiac insufficiency type of death.

November 28, Dr. Christian: The first patient (P. B. B. H. Med. No. 7115) showed in an X-ray taken on November 8, an area of wedge-shaped consolidation with the apex of the wedge toward the hilum. He continued to cough up bloody sputum. Repeated examination of this failed to show any tubercle bacilli but numerous streptococci were present. The liver continued to enlarge and it seemed as if the diaphragm was pushed up quite high on the right. Exploratory puncture, however, in this region yielded no pus. The patient continued to grow progressively weaker and died on November 22 without any additional symptoms developing. Permission for autopsy was refused.

The second patient (P. B. B. H. Med. No. 7427) progressively lost ground without developing any other signs or symptoms of embolism or infarction except blood in the urine, and died on November 14. Autopsy showed acute, large, friable vegetations on the aortic and mitral valves with destruction of part of the aortic cusps. There was a slight old thickening of the mitral flaps with thickening and shortening of the chordæ tendineæ. Infarcts were found in the spleen and kidneys.

## AURICULAR FIBRILLATION

By ROSS V. PATTERSON, M.D.

Assistant Professor of Medicine, and Physician-in-Charge, Department of Electrocardiology, Jefferson Medical College, Philadelphia

---

GENTLEMEN: The clinic hour this morning will be devoted to a study of the cardinal features of the most important mechanical derangement of the heart which produces both cardiac and pulse irregularity. I refer, of course, to the clinical entity which has come to be designated by the term Auricular Fibrillation. The practical importance of this condition in cardiac disease is one upon which emphasis should be laid. Not less than seventy per cent. of the patients admitted to the medical wards of this hospital, exhibiting the symptoms of myocardial insufficiency and circulatory failure, are the subjects of this derangement; if, in addition, there is marked cardiac irregularity, fibrillation of the auricles is almost certainly present. It will not be without profit, therefore, if we bring into review important facts regarding the essential nature of this derangement, the mechanism of its production, its effects, recognition, and distinction from other cardiac irregularities which may be confused with it, together with a discussion of its treatment.

### MECHANISM OF NORMAL CARDIAC CONTRACTION

Since, in order clearly to comprehend derangements of the cardiac mechanism, an intimate knowledge of the normal cardiac contraction is a first essential, I desire to direct your attention to a simple diagram which, in a purely schematic way, represents important anatomical and histological structures concerned in the production of a normal heart beat (Fig. 1).

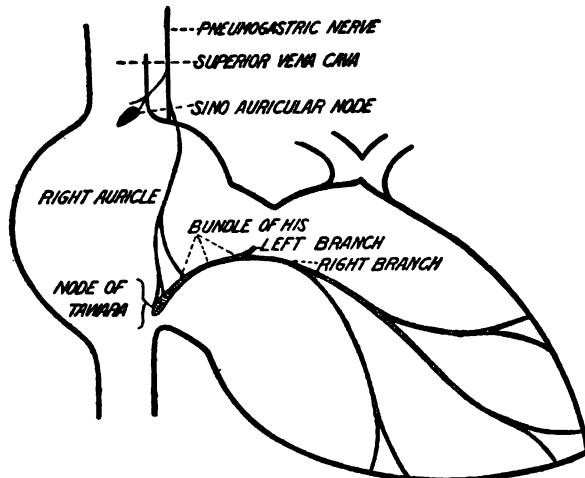
### STIMULUS PRODUCTION

The stimulus upon which each normal heart beat is dependent has its origin in a small mass of primitive cardiac tissue (sinoauricular or sinus node) situated at the junction of the superior vena cava

and the right auricular appendix. The activity of this node may be either augmented or retarded by various influences—mechanical, thermal, toxic or nervous in character, the last named being chiefly effective through fibres supplied by the pneumogastric nerve. While important from other standpoints, these causes of variable function of the sinus node require no more than passing notice at this time, and may be dismissed without further comment.

Under entirely normal conditions, the rate and sequence of the heart beats are determined by the generation of stimuli in the sinoau-

FIG. 1.



ricular node. It has, therefore, been appropriately designated the "pacemaker" of the heart. It not only sets the pace, but also determines the rhythm or regularity, since each stimulus which excites a cardiac contraction requires a definite length of time for its preparation; under conditions which are constant, the required period of time is the same, so that contractions will follow each other with perfect regularity.

Normally, stimuli are sent forth from the sinus node in evenly spaced waves at the rate of about seventy-two per minute, a given wave being separated from the preceding or following wave by a period of  $0.8 +$  second, this period being both the stimulus preparation time, and the time occupied by the succession of events initiated by the

sinus node and constituting a complete cardiac cycle. Stimuli which originate at other than the sinoauricular node are abnormal, and produce disturbance of the cardiac mechanism.

#### CONDUCTIVITY AND THE CONDUCTION SYSTEM

It must be clearly understood that the stimulus wave, and the contraction wave, are the results of the exercise of separate and distinct functions of the myocardium, although the relation between them is an intimate one. The origin, preparation time, and manner of dissemination of the stimulus wave will, in large measure, therefore, determine the rate, rhythm and character of the heart's contraction, since they represent the cardiac response to the stimulus wave, normally originated at the sinoauricular node, and disseminated from this small mass over the walls of the auricles by direct and uniform extension (function of conductivity). The excitation wave is now conducted to the ventricles, not by direct extension over the walls of the heart, but by way of a special conduction system which constitutes the only pathway by which functional activity may be communicated from the auricles to the ventricles. The origin of this communicating link or functional bridge is a node of tissue (node of Tawara or auriculoventricular node) situated low down in the wall of the right auricle posteriorly; it is continued forward and downward as the bundle of His, beyond which it divides into two main branches, the left penetrating the interventricular septum, so that the two branches come to lie upon either side of the dividing wall; further division and subdivision occur until the terminal arborizations are distributed throughout the interior of the ventricles, and finally communicate directly with the myocardium through the network of cells known as the fibres of Purkinje. We can now trace the propagation of a stimulus wave from its origin at the venous end of the heart, over the wall of the auricles, and from the auricles to the ventricles by this narrow pathway, spreading out by various branches, and producing in the resulting activation of myocardial tissue, an orderly sequence of events known as a cardiac contraction. The importance of a knowledge of the origin of normal stimuli, the manner of their conduction from auricles to ventricles, and the dependence of the ventricles upon the auricles for their stimuli, will be evident in the succeeding dis-

cussion. In addition, you are reminded of the refractory state of myocardial tissue which follows each contraction, as another important physiological fact, accounting for the impossibility to tetanize the heart from excessive stimulation, and the failure of the myocardium to respond to a stimulus until after at least a short period of rest. During this rest period the muscle is said to be refractory, and if the number of stimuli be excessive, the myocardium will respond only to a limited number of them. The outside limit of ventricular response is about 200 per minute, even if the stimuli which reach it exceeds that number.

#### CAUSES AND ESSENTIAL NATURE OF AURICULAR FIBRILLATION

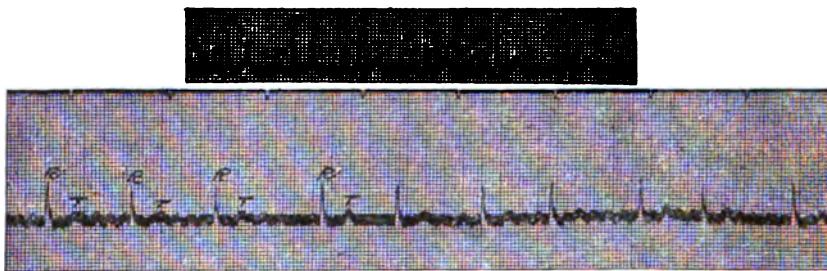
Gross and histological damage to the walls of the auricles may result from a number of causes, and are often of such character as to interfere with normal stimulus production in the sinoauricular node, and with its conduction over the walls of the auricle.

Mitral stenosis, for instance, is an anatomical condition in which, owing to the progressive development of obstructive lesions at the mitral orifice, there is constantly increasing stress upon the walls of the auricle, with dilatation, so that, in extreme grades of this form of endocarditis, we find the auricle over-distended to a marked degree; so much so, in some instances, that its blood content may be several times normal. The result is overstretching of the walls, overgrowth of connective tissue, and more or less dissociation of the muscular fibres.

It is not surprising that such extensive anatomical changes should occasion alteration in myocardial function, and an extreme grade of disorder in the cardiac mechanism, in which the sinoauricular node, the normal pacemaker of the heart, is superseded in its function, and its control of the cardiac rate and rhythm usurped by multiple abnormal foci originating stimuli. These abnormal stimuli originate in the individual muscle fibres of the auricle which are in relation to the node of Tawara, or from other fibres in indirect communication with this node. The over-distention and muscular fragmentation of the auricle produces a marked perversion of its function, in that it no longer acts as an efficient contracting chamber, but stands in a position of permanent diastole, acting merely as a

reservoir for the blood which reaches it from its tributary veins. Cut off from the influence of the sinoauricular node, the individual muscular fibres, or groups of them, continue to contract as a result of self-generated stimuli. Coöordination of contraction of the whole group of auricular muscular tissue is, however, lost; confusion, disorder, and delirium reign in the auricle. Its function of completing ventricular filling at the end of diastole is entirely lost, and it conduces to embarrassment of the ventricle in a degree proportionate to that failure.

FIG. 2.



**ELECTROCARDIOGRAPHIC CURVES.**—Lead II. The upper, smaller, shorter record is a curve taken from a healthy individual with a normal cardiac mechanism. The three vertical waves exhibited in each cardiac cycle are successively marked *P*, *R*, and *T*. The first small waves of each group marked *P* are the result of auricular activity; the *R T* complexes are the outgrowth of ventricular contractions. A period of total inactivity follows each *T* wave. It will be observed that perfect regularity is present, and that each cycle exactly duplicates every other cycle.

The lower curve is taken from a patient with auricular fibrillation. The notable features, as compared with the upper curve, are an absence of *P* waves, irregularity of sequence and height (force) of the *R T* complex, and the presence of irregular small waves following the *T* waves. The electrocardiograph demonstrates the incessant activity of the auricles, their failure to produce one definite wave just preceding ventricular contraction, and the irregularity produced in the ventricle by the fibrillary activity in the auricles.

An ordinate of 1 cm. indicates a current strength of 1 millivolt. The small vertical scale divisions represent a time duration of 0.04 second. The time which elapses between the beginning of auricular contraction and ventricular activity may easily be determined in the normal record. The coarse fibrillary waves in the lower record cause distortion of the *R* and *T* waves.

A much more important influence exerted upon the function of the ventricles, however, is to be found in the generation of the multitudinous, irregular and abnormal stimuli to contraction, originating near the origin of the auriculoventricular node (node of Tawara), and by it taken up and conducted by the bundle of His, its primary branches, and through their many ramifications, ultimately to reach the walls of the ventricles. If the function of the bundle of His remains unimpaired, the ventricles literally will receive showers of haphazard stimuli, producing a rapidity of rate, an irregularity of action, and a variability of force of contraction which tax to the limit the capacity of the ventricles. Owing to a refractory condition

of the ventricles, many of the stimuli will altogether fail to excite them to contract. Others will excite the ventricles to a feeble contraction, but, owing to a short rest period, the contraction will be only of sufficient force to produce a very small pulse wave; while in still other cases, the contraction will be so feeble, and the amount of blood contained in the ventricle so small, as to produce no pulse wave whatever, the ventricle contracting, but not with sufficient force to overcome the pressure in the aorta, and therefore to raise the aortic cusps.

The general circulatory effects in extreme grades of this disorder are a lowering of arterial pressure with a tendency to its transference over to the venous system through the pulmonary circuit, congestion of the lungs, engorgement of the entire venous system, with general visceral congestions; often the outpouring of the fluid portion of the blood into the subcutaneous tissues and serous cavities of the body. Auricular fibrillation, therefore, produces marked disorder of the cardiac contraction, circulatory failure of advanced degree, and, owing to the congestion of the viscera, interference with the function of many organs, the function of the lungs being early and severally affected.

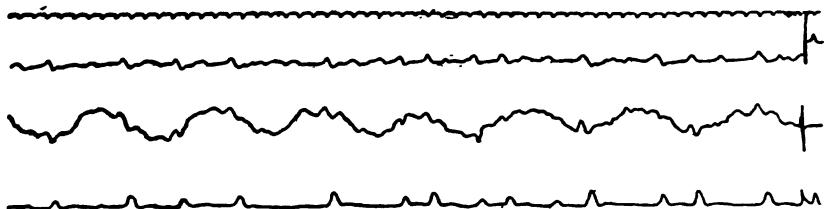
I have so far referred only to mitral stenosis as a cause of auricular fibrillation. I have mentioned it first because the association between these two conditions is most frequent; and also because a study of the effects of mitral stenosis upon the auricle tends to make clear the actual anatomical condition present in auricular fibrillation. It occurs also, however, in mitral insufficiency in which valve defect the ultimate effect upon the auricle is in many respects similar. In the absence of valve lesions with relative insufficiency, the same factors become operative; in a different way, primary myocardial degenerations affect the walls of the auricle, leading to interstitial sclerosis and resulting blockage of the function of conduction along the walls of the auricle and abnormal multiple stimulus production. Dilatation with loss of tone may produce a similar result. Aortic disease, general arteriosclerosis with cardiac hypertrophy, sclerotic and fatty hearts, and granular kidneys, are less frequent causes. Any direct or indirect cause of hypertrophy, myocardial degeneration, or chronic cardiac stress may result in the disordered mechanism which I have endeavored to make clear to you. A consideration of the associated

causal conditions makes it obvious that the incidence of this condition is progressive with an advance of years, and that as males are more often the subjects of the primary conditions than females, with the exception of mitral stenosis, it is more often found among men than among women.

#### SYMPTOMS AND SIGNS

The ordinary symptoms of auricular fibrillation when the condition first comes under notice, are usually those of rupture of compensation, or circulatory failure of less marked degree. These are the symptoms due to the transference of blood pressure from the

FIG. 2.



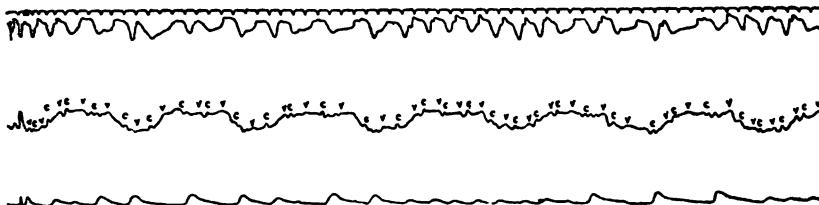
**AURICULAR FIBRILLATION WITH MARKED CARDIAC FAILURE.**—Each time division in this and subsequent tracings marks 0.2 second; the upper curve is the apex beat; middle curve, venous pulse; lower curve, radial pulse. The patient exhibited circulatory failure of marked grade, and died less than twenty-four hours after the record was taken. The autopsy showed auricular dilatation, chronic myocarditis and diffuse chronic nephritis. There were no marked lesions of the valve leaflets. The wholly and continuously irregular character of both the heart action and pulse beats are clearly evident. The cardiac rate is 160; pulse rate 85; the pulse deficit, therefore, representing the incomplete ventricular systole, is 75 per minute. The heart contractions which fail to produce a pulse wave are easily seen. At one place in the record, six successive heart beats produce only one pulsation (the fifth) at the wrist. This record exhibits an extreme grade of disorder in a patient with rupture of compensation.

arterial side over to the venous side through the pulmonary circuit, with the evidences of marked degree of cardiac irregularity and embarrassment, venous engorgement and visceral congestion. The general symptoms of circulatory failure are familiar to you; we may restrict ourselves on the present occasion to the symptoms and signs peculiar to auricular fibrillation, in order to gain time for their more intimate consideration and study.

We may study an extreme grade of disorder first in order that less marked departures from normal may be understood and recognized. With the symptoms of rupture of compensation—œdema, dyspnoea, and the usual evidences of interference with the functions of the kidneys, stomach and liver—we find a pulse extremely rapid in rate, wholly irregular in character, with beats varying both as to

sequence and force. Auscultation at the apex reveals a similar disorder of the ventricular action. It will be noted in many cases that the number of beats counted at the apex, and the number of beats counted at the wrist, will show a marked divergence, so that the heart

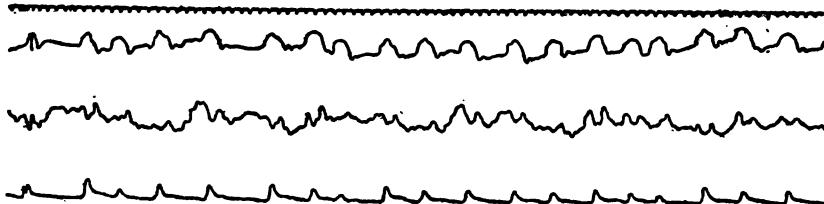
FIG. 4.



**AURICULAR FIBRILLATION.**—Cardiac rate, 120 to 150; recorded pulse rate, 90 to 135; palpable pulse rate, 75 to 100. One run of eight beats is shown in which there is no rest period between beats. By examining the upper record, cardiac contractions producing no pulse wave at the wrist (lower curve) are easily observed at places where the latter waves are relatively long. The *c* and *s* waves in the venous pulse are marked; both are, of course, the outgrowth of ventricular systole. No *a* waves are present since the auricles have ceased to contract coordinately. See Figure 11 for normal *a, c, s* sequence. The multitudinous stimuli originating in the twitching walls of the auricle produce a rapid and disordered action of the ventricles.

beats as compared with the pulse beats may show a difference of as many as fifty, or more, per minute. This is a finding referred to as pulse deficit, and is occasioned by the failure of many weak ventricular contractions to produce a palpable pulse wave at the wrist.

FIG. 5.



**AURICULAR FIBRILLATION WITH NORMAL RATE.**—Complete arrhythmia, the characteristic feature of this disorder, is easily recognized, but is much less evident than in the two previous records. The rate (but not the rhythm) is normal. There is no pulse deficit, every beat of the heart producing an impulse at the wrist. The irregular nature of the pulse might easily be overlooked in such cases, unless the attention of the examiner be specially directed toward its detection. The auricular representative (*c* waves) in the venous pulse (middle curve) is absent, as always, in this disorder, evidencing its failure to act as a contracting chamber.

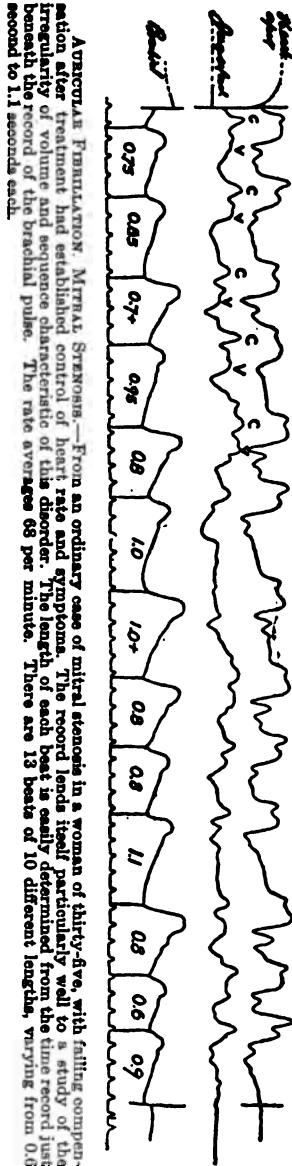
I desire to direct your attention to a series of graphic records which illustrate the character of the disorder better than I am able to describe it. To each is appended a brief note calling attention to one or more important phases of the disorder. A careful study of these will make quite clear to you the essential nature of the dis-

turbance, and will picture to you for the most part that which is felt at the wrist and heard at the apex. The radial pulse in every case shows an absolute irregularity, both as regards volume and sequence. Many of the beats which our instruments of precision will record are not of sufficient volume to be detected by ordinary palpation, so that, for the most part, the radial rates exhibited by the records are above those which could be determined by ordinary palpation. Attention is directed to an occasional absolute failure of a ventricular contraction to produce a pulse wave. The irregularity at the apex is quite as evident as at the radial artery; it is obvious that the intensity of the heart sounds upon auscultation will vary quite as much as the volume of the radial pulse on palpation. The occasional failure of ventricular contraction to raise the aortic cusps, and hence the absence of a second sound following the first sound, together with the disordered sequence and force of contraction, produces a medley of sounds heard in no other condition. The venous pulse shows the absence of auricular waves normally found in such records, since there is, of course, no coördinate contraction of the auricles which normally produces such a wave (see Fig. 11), it being always remembered that in this disorder the auricle has entirely ceased to act as an efficient contracting chamber (see Fig. 2).

It is to be noted that, as a further result of auricular failure, presystolic murmurs dependent upon auricular contraction and presystolic intensifications of diastolic murmurs, likewise dependent upon auricular contraction, will both disappear with the onset of this disorder.

The disordered action of the ventricles is dependent upon the number of supraventricular stimuli transmitted to them from the auricle. If the dissociation of auricular muscle fibres is of extreme grade, each individual fibre in direct or indirect relation to the auriculoventricular node, potentially is a focus of origin of an abnormal stimulus. The stimuli will, therefore, be generated in great numbers. On the other hand, groups of fibres may retain a contraction association, and the functional grouping of the fibres greatly lessen the number of stimuli transmitted to the ventricles by way of the conduction system. Accordingly, the rate will be relatively less. A further modification of the ventricular response may result from interference

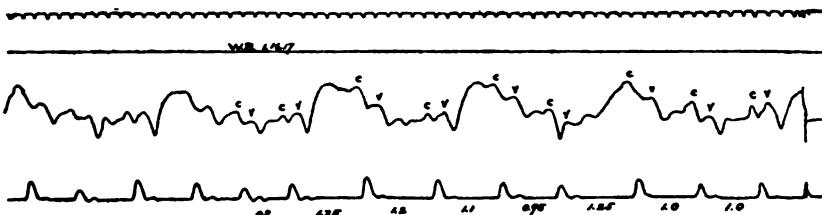
FIG. 6.



AURICULAR FIBRILLATION. MITRAL STENOSIS.—From an ordinary case of mitral stenosis in a woman of thirty-five, with failing compensation after treatment had established control of heart rate and symptoms. The record lends itself particularly well to a study of the irregularity of volume and sequence characteristic of this disorder. The length of each beat is easily determined from the time record just beneath the record of the brachial pulse. The rate averages 68 per minute. There are 13 beats of 10 different lengths, varying from 0.6 second to 1.1 seconds each.

with the conduction function. The bundle of His shares in a liability to damage from those influences which produce sclerosis of the myocardium, fatty change, impaired nutrition, calcareous deposition, and other changes. Various grades of heart block may be induced thereby, producing associated auricular fibrillation and partial heart block. Occasionally the block is almost complete. The ventricular contractions are reduced in number in proportion to the degree of block. To a certain extent the block is beneficial in reducing the circulatory effects attendant upon over-stimulation of the ventricles. A useful therapeutic suggestion is contained in a knowledge of the facts just presented. The thought here thrown out will be developed more fully in the discussion of the treatment.

FIG. 7.



**AURICULAR FIBRILLATION. MITRAL STENOSIS. ADVANCED CHRONIC MYOCARDIAL DEGENERATION. SCLEROSIS OF THE BUNDLE OF HIS. ANGINA.**—Taken from a patient who was under observation and study for several years. The anatomical diagnoses were confirmed at autopsy. There is bradycardia with irregularity as to both volume and sequence. The length of each beat in seconds and decimals shows the variation. The rate was 60 per minute in the absence of cardiac drug effects. The slow rate is due to the sclerosis of the bundle of His, blocking many of the abnormal stimuli originating in the auricle. The absence of waves in the venous pulse such as would be produced by normal auricular contraction (*a* waves) is again notable. During short periods in the course of the case, the pulse rate fell to below 30 per minute (complete block).

#### EFFECTS UPON THE HEART, COURSE AND DURATION

It is, of course, obvious that a heart, the seat of some chronic, progressive, sclerotic or degenerative process out of which the auricular damage has grown, will be seriously embarrassed in the performance of its work by the perversion of normal auricular function, and also by the enormous burden imposed upon it as a result of over-excitation by way of the conduction system. Obviously, the auricular overdistention and disorder is the outgrowth of incorrigible anatomical and histological effects; the condition, therefore, once established, tends to persist. Complete cardiac exhaustion will occur early in extreme cases unless ventricular action is brought within the range of normal rate limits. The outlook is dependent, not only upon the ability to

FIG. 8.

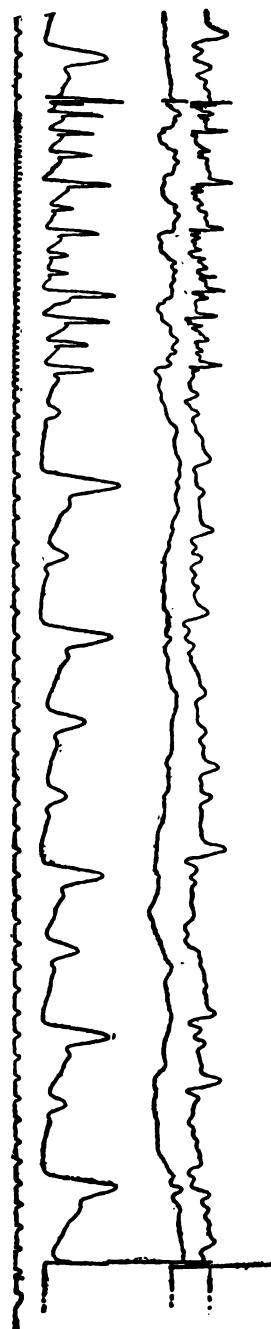
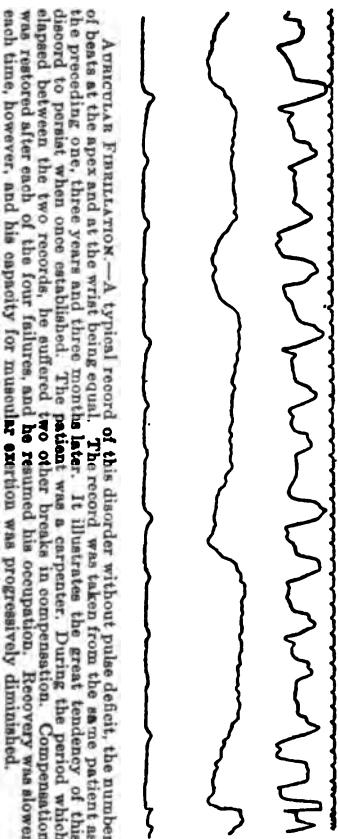


FIG. 9.

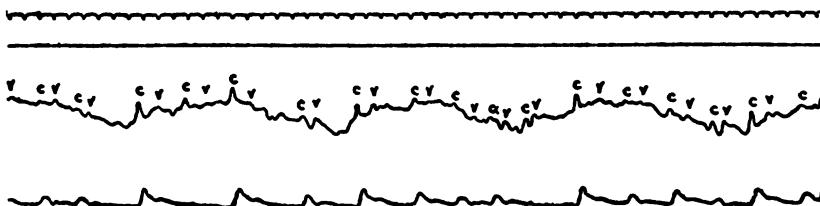


**AURICULAR FIBRILLATION.**—The characteristic irregularity of pulse volume and sequence are clearly evident. It will be observed that the curves have been taken with the recording apparatus running at two different rates of speed. It may be noted that the pulse waves show a tendency to group themselves into pairs or threes. This is usually a result of digitalis, as in the present case, and indicates full effects, and the prompt reduction or discontinuance of the drug. The pulse record somewhat simulates extrasystolic arrhythmia, but differs from an extreme degree of that form of arrhythmia in that there is no tendency whatever to an even spacing of beats, or to an equal duration of similar groups. The importance of differentiation lies in both the treatment and prognosis.

**AURICULAR FIBRILLATION.**—A typical record of this disorder without pulse deficit, the number of beats at the apex and at the wrist being equal. The record was taken from the same patient as the preceding one, three years and three months later. It illustrates the great tendency of this disorder to persist when once established. The patient was a carpenter. During the period which elapsed between the two records, he suffered two other breaks in compensation. Compensation was restored after each of the four failures, and he resumed his occupation. Recovery was slower each time, however, and his capacity for muscular exertion was progressively diminished.

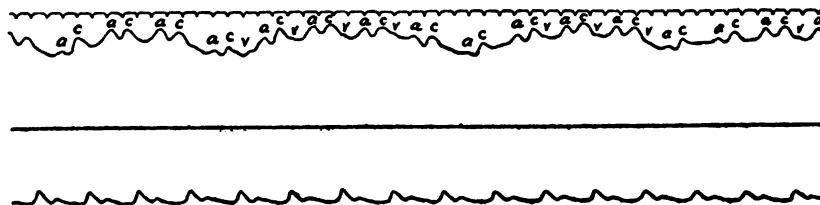
control the ventricle, but also upon the degree of damage which it has suffered as a result of the influence of causes which have also affected the auricle, and perhaps the arteries and other organs as well. Like other cardiac conditions, the prognosis is largely a myocardial question, due importance being accorded to the mechanical and dy-

FIG. 10.



**AURICULAR FIBRILLATION.**—Taken from a patient in whom the condition was intermittent and recurrent. The disorder is clearly evident, but not of an extreme grade. The pulse rate averages 95 per minute. There are occasional incomplete systoles shown by 'c' and 'v' couplets (ventricular representatives) in the venous pulse without a corresponding wave at the wrist. The absence of a wave (auricular representative) in the venous pulse is, of course, due to the over-dilated condition of the auricle and failure of coördinate contraction. It may be assumed that the dissociation of auricular fibres is not of extreme grade in view of the relatively slow rate and the effect of treatment, as shown in the following figure.

FIG. 11.



**NORMAL SINUS RHYTHM.**—Taken from the same patient as the previous record, forty-eight hours later, during which time full doses of digitalis were given. Normal rhythm has been restored. The radial pulse is perfectly regular as to both volume and sequence. The venous pulse shows a wave occurring in each cardiac cycle, evidencing regular, efficient coördinate contraction of the auricles. The normal pacemaker, the sinoauricular node, entirely dominates and regulates auricular activity, and through it and the conduction system, the ventricles as well. Digitalis medication has had little effect in slowing the heart (the rate is approximately the same as before), but here exhibits its effect of restoring lost myocardial tone, and lessening dilatation, in this instance particularly of the auricles. Slowing of the heart requires more prolonged administration. Death occurred suddenly, ten months after the first disturbance of rhythm. During that time auricular fibrillation occurred, and was overcome at six different times; it then became permanent a number of weeks before death, but without marked symptoms of cardiac insufficiency, other than the irregularity, and easily excited breathlessness—evidence of the extreme reduction in cardiac reserve power.

namic considerations affecting the heart, blood-vessels, and their contents. Notwithstanding the incorrigible nature of the auricular, histological and anatomical conditions, much may be done to control the rate; however, in most cases, the disorderly action, while of less marked degree, will persist until the end of the chapter. The outlook in individual cases as regards the immediate future is usually good.

The effects of treatment are often brilliant, and urgent acute symptoms may often be quickly controlled if the essential nature of the derangement which I have endeavored to make clear to you is thoroughly understood, and the plan of treatment which I shall outline for you is applied with discrimination. Many cases go on for many years, though in hospital practice three years is, perhaps, an average expectation. This is, in part, due to the serious damage suffered by the heart before coming under medical observation and direction. Exceptional cases may live for a dozen years, or more.

Rarely, indeed, recovery from the condition takes place, and normal mechanism is restored for a period. I direct your attention to records of a case in which this has occurred. It was the result of the effects of the toxins of diphtheria upon an individual with diffuse nephritis, arteriosclerosis, and myocarditis. With the elimination of the effects of the toxins, normal mechanism was restored. Subsequently, with symptoms of myocardial failure, the condition recurred, with a second recovery. This was repeated several times until ultimately auricular fibrillation became permanent. Death was sudden, and was caused by cardiac failure.

#### THE RECOGNITION OF AURICULAR FIBRILLATION

There is no mystery and no very great difficulty in the recognition of auricular fibrillation. As with other clinical problems, an intimate knowledge of the etiology conduces to a recognition of the disorder, when present. The diagnosis of heart conditions involves an anatomical, functional, and mechanical diagnosis. Auricular fibrillation is the most marked mechanical derangement of the heart. It is associated with various anatomical changes in the endocardium, myocardium and pericardium. Marked degrees of circulatory failure usually bring this disorder to notice. In fact, the added difficulties thrown upon the heart by this derangement are chiefly responsible for the myocardial breakdown, which results in various grades of circulatory failure. Fibrillation of the auricles may be present for a short time without occasioning persistent symptoms of cardiac insufficiency, but even during these few days, shortness of breath on moderate exertion, precordial discomfort, and the consciousness of disorderly or rapid action of the heart described by patients as palpi-

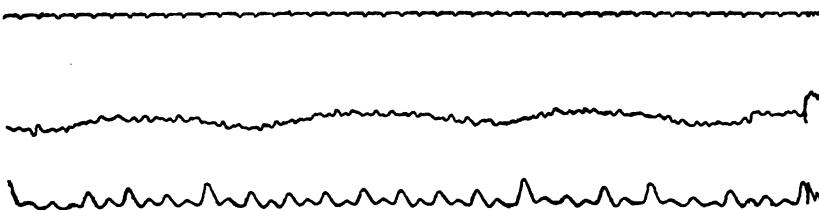
tation, are usually present. In the majority of cases, both the disorder and the incapacity are well characterized. The ordinary symptoms of rupture of compensation are associated with an exceedingly rapid, irregular and insufficient heart action, producing a like irregularity of the pulse beats. I may here take occasion to point out the importance of distinguishing between heart rate and pulse rate. It not infrequently happens that the heart rate will exceed the pulse rate by forty or fifty beats per minute, although the irregularity is marked at either place of examination. The irregularity is complete. There is no other mechanical derangement of the heart in which, with a rapid rate, both the heart and pulse are markedly irregular. This form of cardiac arrhythmia is to be distinguished from all other arrhythmias in that, with an increase in rate, the disordered action becomes more evident. It is, however, evident under all conditions. The pulse deficit, representing the difference between the number of beats which may be palpated at the apex and the wrist simultaneously, tends to disappear with a decrease in rate. The irregularity will become much less evident with a reduction in rate, but will, nevertheless, be clearly recognized upon critical examination. It not infrequently happens that, as a result of more or less generalized sclerotic processes throughout the heart and its lining and enveloping membranes, the bundle of His is affected with sclerosis and interference with its function of conductivity. There will be combined partial heart block and auricular fibrillation. The decreased function of the bundle will tend to inhibit the transmission of the multitudinous impulses from auricle to the ventricle, and, therefore, to reduce in proportion to the degree of interference with conduction, the rate of the ventricles. Such an effect, in a general way, is beneficial. In extreme cases, complete heart block may occur. Whether the function of the bundle remains unimpaired or be interfered with to some degree, the ventricular contractions will be irregular, but the irregularity will be less evident in rates of from sixty to seventy per minute, than in those cases reaching twice these numbers. All other forms of cardiac irregularity tend to disappear with an increase in rate; few of them occur with pulse rates beyond 120 per minute, and then only at infrequent intervals. There are very few circumstances in which any irregularity of the pulse having a rate

beyond 120 can be accounted for in any other way. A rapid and wholly irregular pulse, associated with signs of cardiac failure, is always due to fibrillation of the auricles.

#### TREATMENT

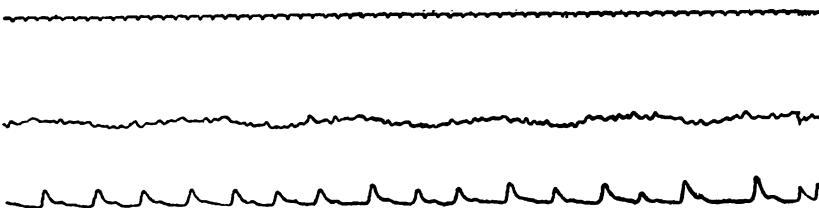
Notwithstanding its seriousness, one may generally assume an optimistic attitude as regards the immediate future of those affected

FIG. 12.



**AURICULAR FIBRILLATION.**—A wholly irregular, rapid-running, low-tension pulse, occurring in a patient who exhibited other marked symptoms of rupture of compensation and circulatory failure. The mechanical derangements are quite evident. The recorded rate is 140 per minute; the palatable rate was considerably less. The venous pulse record evidences the failure of efficient, coördinate auricular contractions, shown by the absence of *a* waves normally found in the venous pulse. Small fibrillary waves (*fff*) reveal the disorder present in the twitching auricular walls, giving rise to multiple irregular stimuli, which, transmitted to the ventricles, produce irregular, rapid and variable force of contraction. The disordered heart action, loss of tone, rapid rate, low tension pulse, venous engorgement, visceral congestions, oedema, and widespread functional derangements due to cardiac failure, indicated the administration of digitalis in full dose. Note effect in following figure.

FIG. 13.



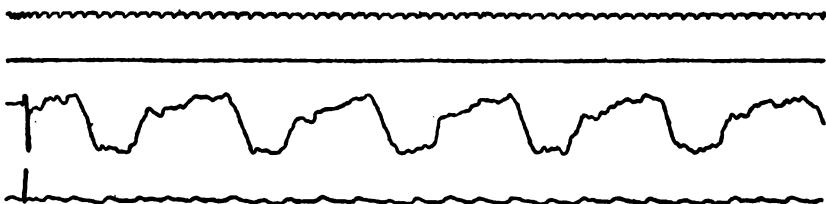
**DIGITALIS EFFECTS IN AURICULAR FIBRILLATION.**—The record was taken from the same patient as the preceding one after the administration of tincture of digitalis M xiv per diem for four days. The cardiac response to digitalis has been prompt and marked. The rate has fallen from 140 to 70 per minute; variations in volume and sequence are slight, but unmistakably reveal the persistence of the mechanical derangement. Reduction in the pulse rate has been chiefly accomplished by the specific depressive effect of digitalis upon the conductivity of the bundle of His. Further depression is not desirable; the effect may persist for some time without medication, or smaller doses may be indicated. Drugs of the digitalis group are often administered beyond the time when they have exhibited their best effects, to the very great detriment of the patient's condition.

with this derangement. There is no condition in which the young physician may so easily gain a reputation, if he will but familiarize himself with the principles of treatment, and administer remedies with judicial discrimination. It is due to its effects in this condition that digitalis owes its reputation as a wonder worker.

The ordinary measures for the restoration of the circulatory balance by the institution of such measures as rest, depletion by purges, venesection, and the treatment of special symptoms due to visceral congestions and venous engorgement, should, of course, be instituted. I shall do no more than mention these measures, however, since I wish particularly to focus your attention upon the principles underlying the use of digitalis and its congeners, in order that the indications for their administration and their proper effects may be clearly understood.

Digitalis, or an allied drug, should be administered in every case of auricular fibrillation in which, while the patient is at rest, the

FIG. 14.



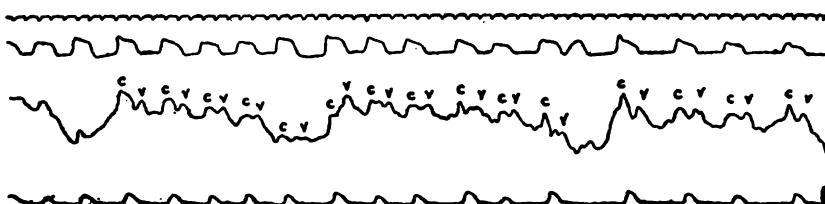
**DIGITALIS EFFECTS IN AURICULAR FIBRILLATION WITH RUPTURE OF COMPENSATION.**—The first of three records presented to show the effects of digitalis on a patient in whom the desired results were not achieved until after a prolonged period of administration. The record shows a rapid, running, irregular pulse of small volume and slight force beating at the rate of 130 per minute. The cardiac apex, owing to dilatation, was not sufficiently forcible to make a record. The respirations are shown to have been 28 per minute. Other signs of cardiac inadequacy were marked. It is clearly evident that the known effects of digitalis upon the cardiac mechanism would result in benefit to cardiac action and improvement in the circulation.

heart rate exceeds 100 beats per minute. It exerts its beneficial effects chiefly by exercising a specific depressive effect upon the function of conductivity of the bundle of His, thereby inducing a degree of partial heart block so that the number of stimuli transmitted from the auricle to the ventricle is reduced until the ventricular rate is brought within normal limits. As soon as this result is accomplished the use of digitalis should be either discontinued, or greatly reduced in amount. Drugs other than digitalis may be used; as a rule, they are all inferior. Those that are useful affect the mechanism of the heart in exactly the same way as does digitalis.

The treatment, therefore, consists in the administration of a drug of this group which will, in the manner indicated, bring the heart rate under control, and within normal rate limits. Let me again say that I refer to the heart rate, and not pulse rate. Personally, I do not

attach as great importance to the choice of the pharmaceutical preparation of digitalis as do some of my confreres. There is more wisdom to be shown in a knowledge of the principles underlying the use of

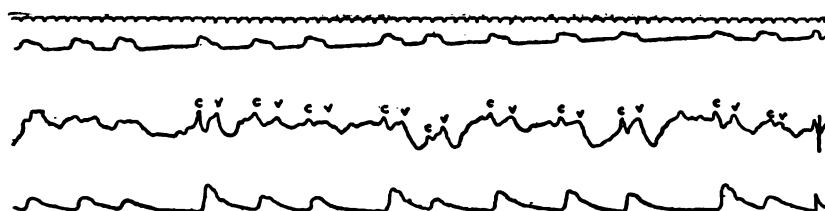
FIG. 15.



**DIGITALIS EFFECTS.**—No. 2. The record shows the effects of eleven days' digitalis administration upon the same patient from whom the previous record was made. The pulse rate has been reduced to 90 per minute. Its volume is perceptibly increased. Increased tone produced in the myocardium has lessened the cardiac dilatation, and increased the force of the contraction so that the apex beat now makes a satisfactory record. It will be observed that there is no pulse deficit. The respiratory rate was 15 per minute. The disorder, still evident but of much less marked degree, is that produced only by fibrillation of the auricles. Digitalis was continued with the expectation of further improvement.

the drug, than in the selection of its form. For the most part, I make use of a good preparation of the tincture of digitalis. In cases in which the disorder is extreme, and the indications are quickly to bring the heart under control, I give as much as a dram of the

FIG. 16.



**DIGITALIS EFFECTS.**—No. 3. The results of twenty-five days' continuous digitalis administration are shown. The pulse rate has been reduced to 60 per minute; the volume has further increased, and the force of the cardiac contractions is presumably augmented. Occasional "digitalis coupling" is shown. Further administration would exceed safe limits. Hope of restoring normal sinus rhythm must be abandoned; the cardiac mechanism may be, in part, beneficially controlled, and ventricular exhaustion, occasioned by overstimulation from the fibrillating auricles, prevented by maintaining the depression of the activity of the bundle of His by the use of whatever amount of digitalis is required to hold the ventricular rate within normal limits.

tincture in twenty-four hours, divided into three or four doses; if no effect is observed in four or five days, the dosage may be slightly increased. It is not to be forgotten that digitalis is a poison, and that signs of intoxication from its administration may occur. Such effects may be manifested by a rather sudden development of nausea, vomit-

ing, diarrhoea, headache, and an aversion to the drug. Often, coincidentally, there is pairing or grouping of the beats. These symptoms often indicate that the full tolerance of the drug has been reached,

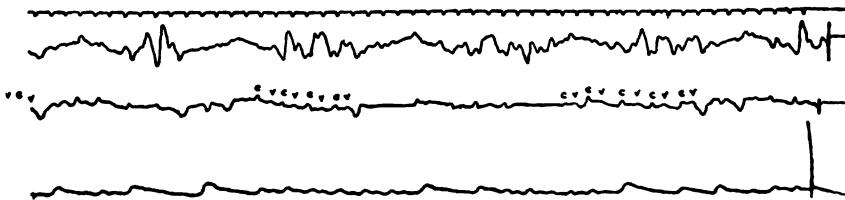
FIG. 17.



**DIGITALIS COUPLING IN AURICULAR FIBRILLATION. PSEUDOBRADYCARDIA.**—The pulse beats occur in pairs without exception, the first being of good volume, and the second much smaller—usually so small a wave as to escape detection at the wrist. The ordinary count, therefore, indicated a heart rate of slightly more than half the actual number of beats. Auscultation revealed a third sound, occasionally a third and fourth sound, after the strong "lub dub" of the first strong beat of the pair, simulating a gallop or canter rhythm. The indications in such cases are promptly to discontinue cardiac medication. The heart rate shown is 65 per minute; the number of beats at the wrist which could be counted at the bedside is 36 per minute.

a condition of saturation developed, and with their development there is a rapid fall of pulse rate to within normal limits. Digitalis administration should then be discontinued until toxic effects have disappeared. The heart rate should be carefully watched, and if, after

FIG. 18.



**AURICULAR FIBRILLATION. MITRAL STENOSIS. MARKED CARDIAC FAILURE.**—The record is the first of two presented to show untoward effects of overtreatment. The second immediately follows. The present figure shows the disorder present at the beginning of the treatment course.

Heart rate	.....	180 to 185
Radial rate (recorded)	.....	140 to 160
Radial rate (by palpation)	.....	130 to 140

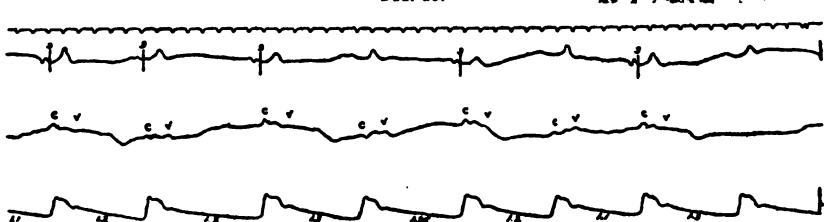
The disorder shown is of extreme grade; the symptoms were urgent. The indications were to restore the circulatory balance and bring the heart under control.

two or three days, with a lessening of digitalis effects, the pulse rate begins to ascend, small doses, sufficient to "hold" the heart at about the normal rate, should be given. In an extreme grade of disorder, full digitalis effects may be obtained in from four to twenty days—most often at or near seven days. With a few days' total discontinu-

ance, it will usually be found necessary to administer lessened amounts.

The rest period in bed should be prolonged beyond the relief of symptoms, in order that the overworked ventricular myocardium may regain its tone, and the function of other organs restored. With the patient out of bed, the amount of digitalis which he will require must be determined by careful study. Most individuals will need digitalis continuously or intermittently for the rest of their lives. The amount varies. In some cases as little as five minums of the tincture per

FIG. 19.



**AURICULAR FIBRILLATION, BRADYCARDIA. EXTREME STROPHANTHUS EFFECTS.**—Irregularity of the pulse in auricular fibrillation becomes less evident with a reduction in the rate, but cannot be overcome. The record presented is from the same patient as the previous one after twenty-four days' strophanthus medication. The length of each pulse wave is stated in terms of seconds with decimals given. The rate had fallen from 180 to 50 per minute; the acute symptoms had entirely disappeared. The record indicates that medication had been pushed beyond the danger point. Notwithstanding the indications clearly present, misunderstood orders resulted in further medication. The hypodermic injection of strophanthin gr. 1-250 was followed by alarming symptoms of collapse, syncope, pallor and cyanosis, cold, clammy extremities, infrequent heart action, beating irregularly for a short time below 20 per minute.

The explanation of the heart action and symptoms induced thereby lies in the recognition of the liability of digitalis, strophanthus, and similar drugs to depress the function of the bundle of His and suddenly produce a complete heart block, with temporary ventricular standstill, and the establishment of a ventricular inherently generated rhythm with difficulty. Unless the ventricle is able promptly to generate its own stimuli to contract, death will occur. Such effects probably account for some of the treatment deaths in cardiac disease. Cardiospasm is probably an additional factor in some cases. In the present instance the use of atropine and nitroglycerine was followed by the disappearance of the alarming symptoms, and the patient made a complete symptomatic recovery.

diem will be all that is required. In other cases, this amount three times a day, or even a larger quantity, may be indicated. Patients themselves, if intelligent and properly instructed, will, under the guidance of their physicians, often solve the problem very well for themselves. Their activities should be restricted to whatever degree may be necessary to bring them within the limits of a very much lessened cardiac capacity. Their subsequent general management is, of course, of the greatest importance, but its discussion is beyond the limits which we have fixed for ourselves this morning.

## **FRACTIONAL GASTRIC TUBE—THE TECHNIC USED IN VARIOUS GASTRIC PROCEDURES**

**By MARTIN H. REHFUSS, M.D.**

**Philadelphia, Pa.**

---

I HAVE received so many inquiries regarding the use of the fractional gastric tube that it seemed to me the simplest solution of this problem would be to present concisely the methods employed with this instrument and the advantages it offers in all forms of gastric work. These I have enumerated under the following subheadings:

### **PRINCIPLES AND DESCRIPTION OF THE FRACTIONAL GASTRIC TUBE**

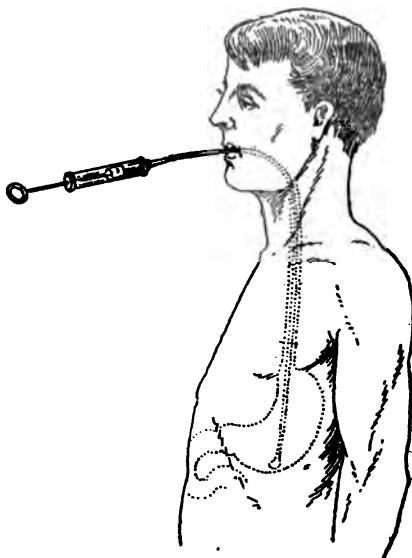
The ideal tube for stomach work must fulfil the following requirements: (1) It should be introduced in the natural way in which substances are ingested, namely, by the act of swallowing rather than by propulsion, as was used with the old tube; (2) it should be of such a calibre that it can be retained in the stomach for varying intervals with practically no discomfort; (3) it should be of sufficient calibre to enable the withdrawal of a representative sample of the gastric contents; (4) it should be of sufficient weight to seek the most dependent portion of the stomach by gravity.

All these points are embodied in the fractional tube. The tip is so constructed that it is easily swallowed and the perforations of the tip are of the same diameter as the bore of the rubber tubing, which should be glass moulded. Anything which will penetrate the tip will go through the tube. In earlier communications on this subject, I pointed out the impossibility of determining anything regarding the evolution of digestion by the old method of the one hour examination. Since that time I have performed many thousands of intubations with the fractional tube and have instructed a number of men in its use.

## INTRODUCTION OF THE TUBE

The fractional gastric tube is swallowed and not pushed into the stomach. The ideal way is simply that of swallowing the tube as if it were an article of food. I have seen many persons simply take the tube in their hand, lay the tip on the tongue and swallow the tube without any assistance. This is the method commonly employed by those who have become more or less habituated to the use of the tube. Most people, however, have a peculiar antipathy to the swallowing of any foreign substance, and the lay mind is beset with the idea that the passage of the stomach tube is a difficult thing. One thing is clear, unless there is obstruction any difficulty occasioned by the swallowing of the fractional gastric tube is purely psychic.

FIG. 1.



Tube in position.

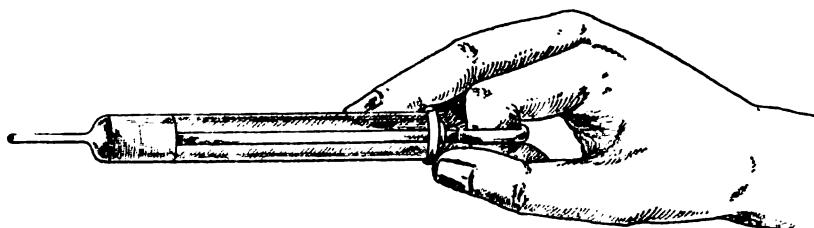
There are three methods by which the tube is introduced, but as I have already said, the mechanism by which it is introduced into the stomach is essentially that of swallowing and not of active propulsion on the part of the operator. These methods are as follows:

(1) The tip and tube after sterilization are lubricated with olive oil, vaseline, glycerine or liquid petrolatum, and after the patient

has removed any false teeth or other movable objects which may be present, he opens his mouth without protruding his tongue. The physician then grasps the tip between the thumb and forefinger and balances the tip with the forefinger on the tongue. With the forefinger the tip is gradually passed along and over the root of the tongue until it engages in the pharynx, after which active swallowing movements on the part of the patient result in the passage of the tube to the stomach. By this method no liquid is taken and the material removed is the pure, undiluted gastric secretion.

(2) The second method consists in passing the tip into the post pharyngeal space and then allowing the patient to swallow a measured amount of water, which is frequently of great aid in securing the desired result. There are many people, however, who can swallow the tube better without liquid than with the aid of substances which will induce peristalsis. It is my rule with an Ewald meal for instance, to use one-half of the tea of the meal to pass the tube.

FIG. 1A.



Method of aspiration.

(3) Occasionally the pharynx will be so hypersensitive that the application of 2 or 4 per cent. solution of cocaine hydrochloride will be necessary, and I usually make several applications to the root of the tongue and the tip of the epiglottis. Any of the above procedures will then succeed.

(4) Every one has encountered individuals who are so nervous and hypersensitive that the slightest touch to the posterior pharyngeal wall seems to throw the superior constrictors of the pharynx into spasm. These are few and far between. The number is extremely small in which the above procedures fail to achieve the desired result. The use of a strong nerve sedative by mouth over several days will usually overcome this difficulty (Fig. 1). (Fig. 1A.)

## HOW FAR SHOULD THE TUBE BE PASSED?

We are told that the distance of the cardia from the incisor teeth is, on an average, forty centimetres and that the passage of from fifty-five to sixty-five centimetres of tubing results in the introduction of the tube into the stomach. Duodenal intubation requires the passage of more tubing. Any figures are purely arbitrary inasmuch as no definite amount of tubing can be given which will serve as a working guide. I never use measured tubes. Instead, if the patient is a visceroptotic more tubing is passed than in an individual with broad epigastric angle and thick, barrel-like abdomen. I usually place the tip at the umbilicus and run it on the surface of the body to the neck and alongside the neck to the angle of the mouth, in that way roughly measuring the desired amount of tubing. For duodenal intubation I pass enough tubing to go externally on the body from the angle of the mouth to one of the anterior iliac crests. The physician's judgment will therefore largely determine the amount of tubing to be swallowed. It is obvious that to reach the most dependent part of the stomach, no two persons will require the same length of tubing. I have frequently come across tubes which were too short to properly reach the bottom of an elongated or displaced stomach.

## HOW DO WE KNOW THAT THE TUBE IS IN THE STOMACH?

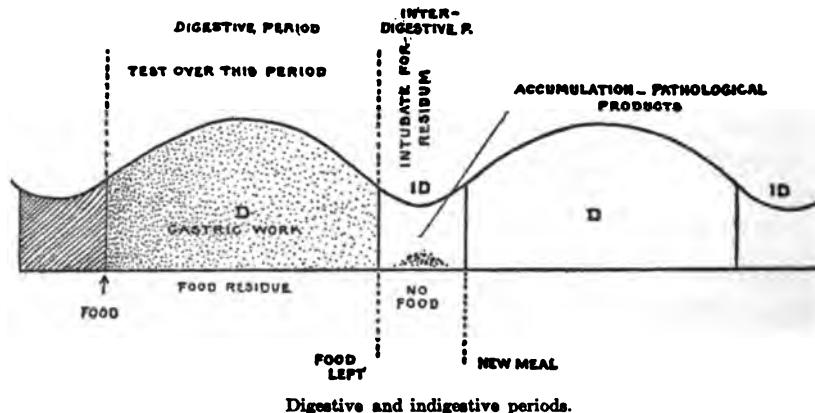
Ordinarily the tip is arrested for a short time just above the cardia. We know that the tube is in the oesophagus and not in the stomach by the following signs: (1) Aspiration results in the removal of a small mucoid "oesophageal sample" or none at all; (2) aspiration soon discloses a block, no further material being aspirated owing to the fact that the oesophageal wall closes around the perforations of the tip; (3) the injection of fluid through the tube occurs readily, but the fluid cannot be reaspirated owing to its passage into the stomach; another point of importance is the passage of the tip into the air chamber before it goes into the fundus. The signs which indicate this occurrence are the following: (a) Aspiration is readily performed, but nothing but air returns; (b) material is readily injected, but nothing but air can be obtained on reaspiration. The normal gastric contents are easily recognized. Inability to obtain them or blocking of the tube is due to the following causes:

(1) The tube is not clean, (2) the tube is in the cardia, (3) the tube is in the air chamber, (4) the tube is blocked by material too coarse to pass through it, (5) too much tubing has been swallowed or the tube may be caught in the pylorus, (6) very occasionally hyperactive peristalsis may tie a knot in the tube. The evidence supporting one or another of these possibilities will usually be apparent to a skilled observer, but they must be borne in mind. A description of some of the uses to which this tube can be put will not be out of place.

**EXAMINATION OF THE INTERDIGESTIVE OR REST PHASE  
(SO CALLED EMPTY STOMACH)**

To make an exact study of the "gastric residuum" the tube must be lubricated and passed without any liquid. There will be usually

FIG. 2.



a slight arrest at the cardia, after which aspiration is readily performed. I cannot insist too strongly upon gentleness in manipulation, in fact, comparatively few people acquire the art of *feeling* the syringe so that any possible strain or undue tension may be detected. I always insist upon performing aspiration with one hand instead of two, grasping the barrel of the syringe with the index and second fingers while the thumb presses on the edge of the syringe as shown in the diagram, and the remaining fingers grasp the piston. In this way but little tension can be exerted, in fact, it is a rule in this work that tension should never be exerted in aspiration (Fig. 2).

It is not surprising that occult blood is so frequently found when technic is faulty. If I found occult blood after the use of the proper tube and syringe I consider it of diagnostic value. For a long time the perforations in the tip were too sharp, but this has been largely overcome. Frequently I have dipped the tip in hot paraffin to coat it and overcome the possibility of slight trauma. After the tube is in place, the residuum is collected by complete aspiration in different positions, recumbent, right and left lateral, because it will be apparent to anyone that only in these different positions will the stomach be completely aspirated.

I consider the study of the empty, or fasting stomach, as one of the most important parts of gastric study. Food retention, bleeding, pus, mucus, and swallowed oropharyngeal or pulmonary material are most readily demonstrated at this phase when frequently no trace of these is seen after the introduction of the test meal.

#### FRACTIONAL GASTRIC ANALYSIS

The technic of this examination is too well known to merit description. In the laboratories of Physiological Chemistry of the Jefferson Medical College, we have demonstrated the utility of this method of examination for studying all varieties of food stuffs, and the data connected with such studies will appear elsewhere. The point, however, is that we are not necessarily confined to the use of the Ewald test meal, we may use broth, the water meal, crackers, milk, a mixed meal, in fact, anything. Under the direction of Doctor Hawk, every variety of food stuff has been tested and these studies will shortly appear in published form. Suffice it to say, however, that practically any form of food, if properly prepared, can be tested in this way.

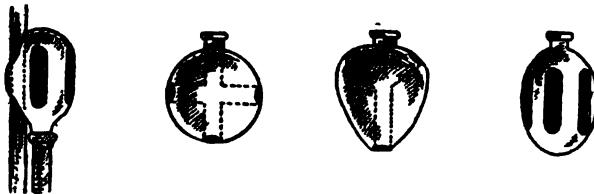
I have been frequently asked as to the intervals of testing. The fifteen-minute intervals are purely optional and any interval may be used. In studying the response to stimuli, we may aspirate as often as every minute, for the first few minutes, and in pyloric stenosis I frequently aspirate at hourly intervals. The fifteen-minute interval is convenient because any noteworthy change is readily recognized in these intervals. If there is a marked digestive delay half-hour

intervals are sufficient. In our studies on the neutralization of the gastric secretion by the Sippy treatment, I employed ten-minute intervals (Fig. 3).

#### THE DETERMINATION OF THE END POINT OF FOOD DIGESTION

There is a distinct difference between the exit of all food from the stomach and the transition of digestive to fasting secretion. In many instances after all food has left the stomach, a post-digestive hypersecretion will continue at times for hours. However, the ordinary case will present the following points at the end of food digestion: (1) No more food can be aspirated, (2) the end of digestion is usually marked by a change in the appearance of the samples and some admixture of pyloric mucus, (3) the injection of water into the stomach and lavage will reveal the absence of all food, (4) the

FIG. 3.



Tips used with tube.

injection of air produces a hiss instead of the gurgling sound heard when there is liquid in the stomach. If the tube is introduced to sufficient length, the simple disappearance of food from the sample aspirated will usually mark the end of food digestion in the stomach.

#### LAVAGE OF THE STOMACH

The fractional tube offers a convenient form of gastric lavage, especially when performed on the fasting stomach in the morning. Except with pronounced retention or stenosis I never use the old tube. By means of the fractional tube all material can be removed from the stomach and then lavage performed in one of the following ways:

(1) The lavage liquid can be injected through the tube and then reaspirated. That this offers an efficient method of lavage will be

evident from the fact that the samples change as the lavage accomplishes its purpose.

(2) The patient may drink water and aspiration follow. In other words the swallowed water represents the lavage water.

(3) Another method, after complete evacuation of the stomach, consists in the use of either of the above methods followed by siphon, a long tube being used and the end of the tube allowed to drain in a basin on the floor.

Lavage is indicated for the removal of mucus, the cleansing of the stomach in gastric carcinoma and infected gastritis, and for the removal of gastric retention. Water, salt solution, alkaline solutions, or the use of diluted solution of liquor antisepticus alkalinus is indicated when mucoid material proves particularly rebellious. In fact, I know of no more efficacious method of removing mucus than by means of the fractional tube. I have frequently removed large amounts of mucus with the fractional tube when the syphon lavage with the older tube failed to remove it. This is due to the fact that traction on the mucus is more easily performed with the small tube and syringe.

Lavage has been used in the study of gastric cytology. Here the use of the fractional gastric tube is practically the only satisfactory method which we have. The tip is coated with a thin layer of paraffin and lubricated and the residuum very gently removed. Then successive increments of weak physiological saline solution are injected and either reaspirated or, better still, allowed to syphon off, a long tube being used. If force is used in injecting and reaspirating the salt solution, slight trauma may occur and even small pieces of the upper mucosa may be found (erosive gastritis, etc.). I have noted these, but I do not believe they should exist as a separate entity. The material can then be centrifugalized and examined in the fresh condition or properly fixed and stained. The flora of the ordinary resting stomach is usually so rich from swallowed material that this method of examination has lost its value as a routine procedure.

#### APPLICATION OF MEDICAMENTS

The fractional tube offers us a convenient way of applying strong medication directly to the gastric wall, thus *establishing a form of direct gastric therapy*. All medication which is destined to act on

the stomach must necessarily be limited in its nature owing first to systemic and side effects, and secondly to its admixture and dilution with the gastric contents both in the resting and the fasting stomach. *This means that direct local medication, sufficient to have a pronounced effect, cannot be administered, owing both to its systemic absorption and to the dilution which it must of necessity undergo, the stomach being never empty.* The use of strong silver solutions, the nitrate, iodide, argyrol, protargol, ferric chloride, and even iodine, was, heretofore, impossible. (The last one I found to have irritative effects on the stomach.)

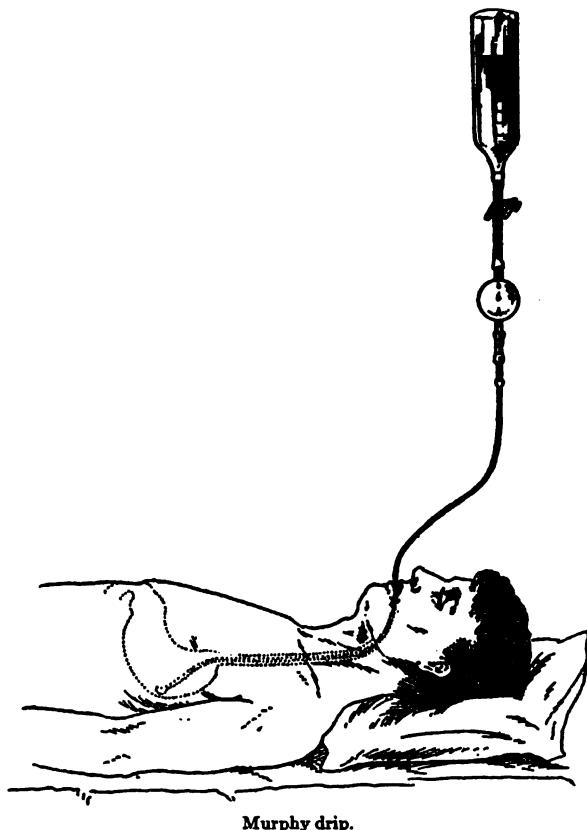
Therefore to obtain direct local action we must first, completely empty the stomach, secondly, apply the medicament, thirdly reaspire the substance before systemic or dangerous side action can occur. The silver group can by this means be applied directly to the mucosa and after five or ten minutes either neutralized with various solutions and reaspirated or simply reaspirated, thus offering us a means of direct therapy of particular value in gastric infections and the various forms of gastritis. By this means we can localize our therapeutic effects to the stomach and obtain *direct gastric therapy*. In cancer of the stomach, for prophylactic purposes, the application of disinfectant aqueous and oily suspensions to the stomach is particularly of value.

#### THE USE OF THE MURPHY DRIP WITH THE FRACTIONAL TUBE

Jutte described transduodenal lavage, but the fractional tube, both in the stomach and in the duodenum, when connected with the Murphy drip, offers a number of advantages. We can obtain the continuous application of medicated solutions to the gastric wall and to the upper intestinal tract. This is now a routine form of treatment with me in many conditions. I have treated gastric hemorrhage by the Murphy drip and fractional tube with 1:1000 ferric chloride, with adrenaline chloride 1:1000 (1/2 dram to the quart); duodenitis and gastritis with argyrol and protargol by drip; gastritis, gall-bladder and duct disturbances, where duodenal cultures gave evidences of infection, by means of these substances with the duodenal tube. The use of urotropin, sodium sulphate and sodium bicarbonate, with the drip, is indicated in gall bladder conditions. In cases of intestinal stasis with evidences of trouble in the upper small bowel

and biliary region, I have used, where possible, the Hayem formula of three sodas: sodium sulphate, phosphate, and bicarbonate, followed after the drip is completed by the injection of castor oil through the tube. The possibilities of this form of therapy, not merely for

FIG. 4.



Murphy drip.

medicinal but dietetic purposes, has strongly appealed to me. Prepared and predigested foods can also be readily administered in this way (Fig. 4).

#### DELIMITATION OF THE STOMACH BY MEANS OF FRACTIONAL TUBE AND AUSCULTATION

With the fractional tube in place the injection of air into the stomach through the tube is followed by a variety of sounds from

the standpoint of auscultation. If the stomach is full of material, the tube is in place, and ~~can~~ be injected with a bulb, auscultation will reveal—depending on the type of the material—large gurgles; if little material liquid in character is present, the gurgling sounds are more rapid while if the stomach is almost empty, the sounds will resemble sticky râles. If the stomach is entirely emptied, simply the hiss of rushing air will be heard. The sounds are best heard over the area of the stomach and fade away as the bowl of the stethoscope passes the border of the stomach. By means of the bulb and tube the stomach can be inflated and readily outlined by percussion or even outlined by the character of sounds heard by the inrush of air through the tip of the tube. After the stomach is evacuated, this procedure takes but a few minutes and often yields data of importance.

#### DETERMINATION OF THE VELOCITY OF THE FORMATION OF SECRETION

In our studies on the psychic secretion, it became apparent that the secretion was formed more or less constantly and that for a given time interval the quantity of secretion formed in response to a certain stimulus varied greatly. With the tube in place, the stomach was completely emptied at a certain time interval and then, after a definite, fixed time, the stomach was again completely emptied. We found that the amount of secretion formed in fifteen-minute intervals varied all the way from a few cubic centimetres to over one hundred. In fact, in digestion hypersecretions, and in continuous hypersecretions, the complete withdrawal of the hypersecretory liquid is often followed in a remarkably short time by the appearance of rapidly formed secretion. This question is worthy of more serious study, and the fractional tube offers an easy method for its determination.

#### SYPHON METHOD OF EXAMINATION

With a long tube it is possible to introduce enough tubing to enter the stomach or duodenum while several feet are still protruding from the mouth. With the patient in the recumbent position, the tube can be allowed to drain into a vessel while the material is syphoned off. In obtaining bile, this is the method I always employ after the

bile is once apparent. If a liquid test meal is given the syphon method can be used, as was long ago suggested by Bondi. In the study of hypersecretion also it offers a simple means of obtaining the secretion. Furthermore the presence of occult blood in the syphon specimen is of more value than that from the aspirated specimen, inasmuch as the possibility of trauma is greatly minimized (Fig. 5).

#### THE ADMINISTRATION OF HYDROCHLORIC ACID

It has been suggested to administer hydrochloric acid throughout digestion. This can be accomplished either by fractional injection, or through the Murphy drip and fractional tube after a meal has

Fig. 5.



Syphon method of obtaining gastric secretion.

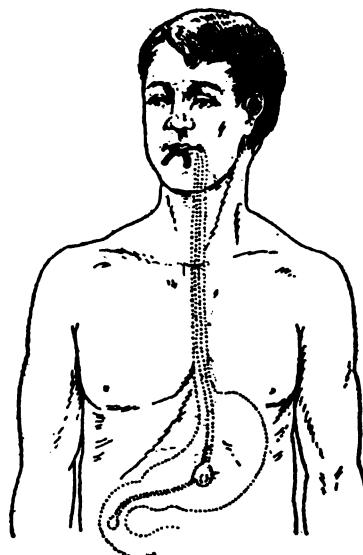
been ingested. In delayed digestion and in the achylia, after the patient has eaten a meal, the fractional tube can be inserted and the dilute acid given at intervals, or as I have done, the Murphy drip apparatus can be connected and 0.25 per cent. HCl allowed to drip slowly throughout the digestive period. In one case of achylia which I reported, the dilute acid was allowed to drip into the stomach throughout the entire digestive period while another tube was inserted into another part of the stomach. From the latter tube specimens were obtained for titration. In that case over 400 C.c. of 0.25 per cent. HCl were injected before the titratable acidity approached the normal digestive figure, and that did not occur until

one and a half hours after the ingestion of the meal. Stronger solutions can be introduced through the tube than can readily be taken by mouth.

THE DETERMINATION OF THE AMOUNT OF ALKALI NECESSARY TO  
NEUTRALIZE THE GASTRIC SECRETION

Sometime ago I made a study of the effect of the Sippy treatment on gastric acidity. I found that in certain cases the doses recommended were more than sufficient to neutralize the acidity, in others

FIG. 6.



Double gastro-duodenal tube in position.

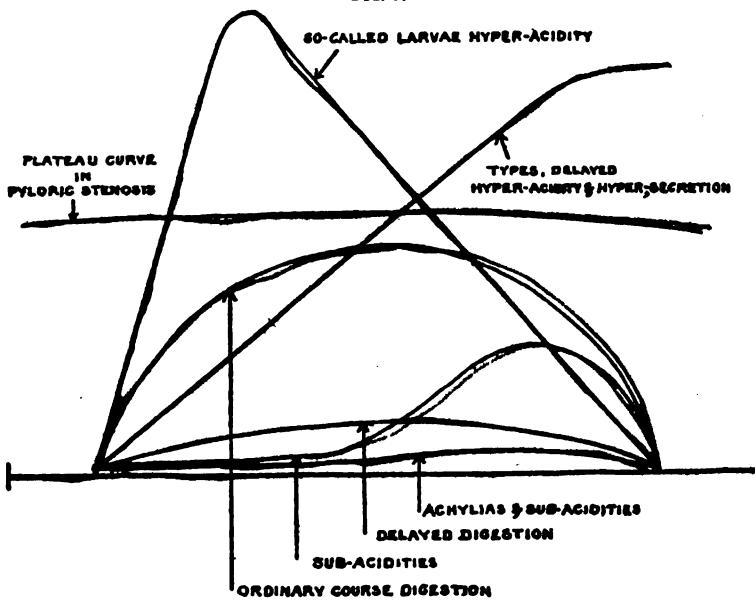
they were not sufficient. It is evident that titration figures alone will not indicate how much of the neutralizing agent is necessary, inasmuch as the volume of the secretion more or less constantly varies and cannot be easily determined. The loss by the pylorus and the gain by the constant formation of new secretion make the variables in the problem so many that no mathematical formula can be deduced for determining just how much alkali will neutralize the acidity. I quickly realized that the only method was to follow out the treatment recommended over several hours, testing at intervals the samples

which were aspirated. I have in this way estimated the effect of the alkali. It is interesting to note, in this connection, the fact that whereas the acidity will be neutralized at times for hours, a sudden divergence will sometimes occur in spite of the exact technic.

#### DOUBLE GASTRIC AND DUODENAL INTUBATION

In the beginning of this study, I passed a duodenal tube into the duodenum, and after the character of the material aspirated assured me that it was in the duodenum, I passed a second gastric

FIG. 7.



Possibilities in the evolution of the secretory curve of digestion.

tube independent of the first. In this way it was possible to follow simultaneously digestion both in the stomach and in the duodenum. Since then I have used a double gastric and duodenal tube of my own construction, in which the gastric tube and its tip were attached by a sliding arrangement to the duodenal tube. This arrangement assures separation between the two tips, the distance of which can be readily adjusted. There is no difficulty in passing this tube—the only trouble is in waiting for the duodenal tube, which is the longer, to enter the duodenum. Both methods are satisfactory. For

simplicity, however, the first method or double insertion, is equally reliable and has the advantage that during the period while the duodenal tube enters the duodenum, the patient has only one tube in his mouth. After this has reached its destination it is an easy matter to insert the second tube.

Double gastric and duodenal intubation is a practical procedure and should yield many points of interest. The most interesting point to me has been the remarkable comminution of food by the stomach and the great difference in appearance between gastric and duodenal samples after intubation. The variations in bile flow are also of interest.

I have attempted to enumerate some of the uses to which the fractional tube can be put. Ingenuity will suggest others, but the above have been the outcome simply of necessity in approaching some of the problems with which we have been confronted in studying the stomach (Fig. 6).

## A LECTURE ON ANEURYSMAL HÆMATOMA

By CHARLES GREENE CUMSTON, B.S., M.D.

Privat-docent at the University of Geneva; Honorary Member of the Surgical Society of Belgium; Fellow of the Royal Society of Medicine of London; Member of the Medical Society of Geneva, etc., etc.  
Geneva, Switzerland

---

**GENTLEMEN:** Aneurysmal hæmatomata, or diffuse traumatic aneurysm, as these lesions are also called, are relatively uncommon in civil practice, but in warfare they are much more frequently met with and it is my intention in this lecture to dwell upon the aspects of this important lesion from the standpoint of war surgery.

Generally speaking, it can be said that an aneurysmal hæmatoma is equal in its physiological results to a ligature of the vessel involved. The artery is interrupted in its continuity and cannot, either by itself or its collaterals, irrigate the portion of the limb below the site of the lesion. The portion of the limb subjacent to the muscular lesion is suddenly placed in the worst possible condition of nutrition—a condition absolutely identical to that resulting from experimental ligature.

An artery can be wounded in many ways and the teaching has been to divide cases into contusion or slight lesions which do not result in a complete solution of continuity of the vessel walls and injuries causing complete division of the artery. Contusions and slight injuries are apt to be less serious than complete division. A contusion or an incomplete section of an artery may result in a circumscribed aneurysm with a distinctly limited sac, due to the absence of solidity of the arterial wall in the area of the injury.

Such an injury with or without the development of an aneurysm may, nevertheless, be the causative factor of very serious complications, among which I would particularly mention the detachment of a clot resulting in an embolus. But this class of arterial injuries I shall leave aside, as they are certainly more frequent in civil practice than are aneurysmal hæmatomata, to which I desire to confine my remarks to-day.

The second class of arterial lesions is above all a matter of warfare pathology, especially bullet wounds of the arteries. The anatomical condition requisite for the production of an aneurysmal haematoma is *the formation of a sac within which the injured vessel is to be found*, and which keeps up and progressively increases the size of the aneurysm the more the loose connective tissue becomes dissociated. Time will not allow me to describe all the possible lesions; they are numerous and you have already found them described in your text books, so that I shall merely point out to you certain points which seem to me of particular interest and worthy of attention.

Your text books will tell you that a true reaction of the connective tissue takes place around the wounded artery; that at a little distance the muscles become adherent to this fibro-connective sheath, which is composed of muscles, aponeurosis and condensed connective tissue. Then deposits of fibrin result from the coagulation of the extravasated blood which increase the resistance of the neoformed walls of the sac, and the process of fibrin deposit may become so intense as to completely fill and obliterate the sac, transforming it into a fibro-connective tissue mass which offers a tendency to undergo regression by absorption.

Such, gentlemen, is the classic description of the process under consideration, but it seems to me rather to incline towards the hypothetic. In reality, two anatomical conditions may be produced. First, the artery is injured in its sheath and the sheath is not injured. The sheath may be sufficiently resistant and therefore becomes distended with blood, compresses the vessel in question and forms a small haematoma within its lumen. The extravasated blood spreads above and downwards in the sheath, but in these circumstances the sac will be very limited in extent. The walls of the latter are *pre-formed*, so that the blood escaping after the injury, to the artery finds an already existing barrier offered by the vascular sheath.

The same process occurs in an inflammatory focus—a circumscribed abscess with thick fibrous walls distinctly limited in extent, in which the artery is enclosed. Now, if this artery should be injured either from a trauma or an ulcerative inflammatory process the blood will escape into a preformed sac, and the result will be a haematoma

with distinctly developed walls. All this is quite like hæmatomata occurring in tuberculous abscesses of the spine or hip, likewise within the lumen of an hydatid cyst. But in injuries of warfare the problem is quite different. Here we have a normal limb with arteries and muscles in a healthy condition because the soldier is still on the easy side of forty-five years of age. The limb is transpierced by a bullet which divides an artery, and a free copious hemorrhage results under pressure between muscles easily dissociated, while the pressure of the arterial blood forcing itself between the muscles quickly dissociates the loose connective filling the intermuscular interstices. On account of these circumstances the blood rapidly fills the portions of the limb both above and below the site of the vascular injury, as no serious obstacle is offered to its progress. This blood infiltration into the intermuscular connective tissue is almost exclusively met with on the posterior aspect of the thigh, along the sheath of the sciatic nerve, between the biceps and semi-membranosus muscle and at the posterior aspect of the leg, between the *gastrocnemius* and *soleus* or between the latter and the flexor muscles.

The increase in the size of the blood collection is only hindered at the time when the distended muscles and aponeuroses offer a resistance equal to that of the arterial pressure. Not until then will the extravasation of blood cease. After having extended far along into all the neighboring intermuscular spaces the blood coagulates, and if the collection is left to itself it is most prone to become a focus of infection and transforms into a vast abscess.

This process may be quite correctly compared to intraperitoneal blood collections resulting from a ruptured extra-uterine gestation sac. If the peritoneum is already the seat of some infectious process, and if adhesions are present, the blood collection becomes encysted in a walled-off pocket. On the other hand, if the peritoneum is healthy there is a free extravasation into the peritoneal cavity.

Therefore, in gunshot injuries of an artery, there follows what may be very properly called a diffuse blood collection in an entire segment of the limb, and this condition has been proven to exist in the many operations which have been undertaken during the present war.

Another interesting anatomic condition to which I wish to direct your attention is the nature of the arterial lesion, which greatly varies

from one case to another, according to the type of projectile causing the injury. When the projectile is sharp and pointed it will produce a cleaner division of the vessel than will a shrapnel bullet, for example, which causes contusion and tears the vessel. Therefore, in these circumstances the artery is contused for quite a bit of its length and not infrequently is torn at several spots. Because of the resulting friability an artery thus injured cannot be ligated and the best that can be done is to catch the ends in haemostats, which are then left *in situ*.

When the projectile remains in contact with a vessel, the resulting lesions are always more serious. It is prone to provoke serious secondary complications, such as ulceration of the satellite vein, if the vein itself has escaped injury, or a secondary injury to some nerve trunk near the vessel.

It is interesting to know that the structures going through the lumen of the aneurysmal sac, filled with clots, are far from being free from injury, although they may not have been directly involved by the projectile. The satellite veins are usually the seat of contusions from the projectile, and, at all events, they cannot easily withstand a prolonged sojourn in the midst of clotted or fluid blood, as they become rapidly macerated, their structure being much more delicate than that of the arteries. When examined, they appear soft and flabby, their walls are friable and tear with ease.

Now, if the haematoma is not incised and cleaned out, the infiltrated veins may rupture and bleed directly into the aneurysmal sac, but this loose venous blood does not increase the size of the collection to any considerable extent, for the very good reason that the aneurysmal tension is usually greater than the venous pressure.

The nerve trunks are similarly infiltrated with serum and lose their normal toughness. They frequently become dissociated, from which arises a disturbance in their functional integrity. The muscles are not very materially involved at the beginning of the process, but if the haematoma is left to itself, they at length lose their tonicity and become limp, distended and dissociated into several bundles of muscular fibres.

The details of the clinical evolution of aneurysmal haematomata have been fairly well described in their essential points in most text

books on surgery, so that there are only some peculiarities which merit your attention, and to which I shall now refer.

The first is that the general evolution of the process seems for sometime to be that of a vast abscess. The entrance and exit openings of the projectile tend to contract and close very rapidly, and, at all events, no longer communicate with the hæmatoma after the lapse of a few days. Occasionally they cicatrize completely, so that the blood collection is completely closed off on all sides.

For several days the collection will rapidly increase, causing a painful and very evident tumefaction. The pain is, in the first place, located in the wound area, but soon extends to the rest of the limb. You must always remember that the bullet may have involved a more or less important nerve trunk, along with the artery, and most frequently it is the satellite nerve of the vessel.

On the other hand, the lumen of the sac is crossed by nerve filaments which become irritated by the blood clots in which they are embedded, so that the ensuing neuralgia is likely to be severe.

A most important point, one which may be likely to put you on the track of the correct diagnosis, from the very commencement of the lesion, is *the rapid increase in the size of the limb, which, when once it has attained its extreme limit, will hardly, if at all, vary in circumference during the weeks following, providing the hæmatoma does not become infected.* This latter occurrence usually takes place, however. Now, you must remember that in warfare the patients are transferred from one medical post to another during the first few days following the receipt of the injury, so that this circumstance is overlooked and when the case comes into your hands you may very likely suspect that the process is a vast pus collection. Therefore, always be on your guard in this matter, but if you bear in mind the possibility of an aneurysmal hæmatoma, you will not be apt to make a mistaken diagnosis.

Another diagnostic sign which may have great importance if noted at the beginning of the process, is *the absence of arterial pulsation in the portion of the limb seated on the distal side of the injury.* Of course the pulsations disappear as soon as an artery is divided and the blood escapes into the surrounding anatomical spaces, and should this be noted before a vast collection has formed this sign is of the

utmost diagnostic value and should always be noted on the patient's transfer bulletin.

However, when a large fluid collection, be it blood or pus, surrounds an artery, the absence or a decrease in the force of the arterial beats is of less import, because these may be the outcome of compression. But a simple decrease is rather more in favor of a collection of pus, although, if the artery is merely wounded but not completely divided, a haematoma is produced and the blood may still pass into the distal end of the injured vessel. Therefore, we here have a presumptive sign, but not one of certitude.

Fluctuation may occasionally be observed, but the focus is in most cases too tense for this sign to be made distinct, and then again, clotted blood is less apt to give rise to the sensation of fluctuation than a frankly fluid collection.

There is yet another sign upon which much hope was based from the diagnostic viewpoint. I refer to the fact that the sac is frequently animated by pulsations felt by the hand when applied flat over the limb. There may also exist expansion, as in circumscribed aneurysmal sacs, but for this sign to be present the collection must be superficially situated, small in size and only slightly tense. It is wanting in very large and tense collections and I think I may say without hesitancy, pulsation and expansion of the sac are most inconclusive signs, always allowing for exceptions to the rule. Let me add that these signs are unreliable, because fluid collections composed of pus have a distinct origin, and, being in no way related to an arterial lesion, may be in contact with the principal artery of the limb, and, should the hydraulic conditions be propitious, it can very readily become the seat of pulsations. Therefore, you clearly see that it may be a matter of extreme difficulty to diagnose an aneurysmal haematoma in its early phases and to differentiate the lesion from an abscess in the phase of congestion, especially if both pulsation and fluctuation are absent.

The anatomical site of the wound alone may occasionally lead one to suspect that an arterial lesion exists, but there is a group of symptoms which possesses, perhaps, greater value in this respect; I refer to *the patient's general condition*. If you have a case of a vast abscess of the thigh in its early stages—in the phase of congestion—there will be a rise in the temperature in the neighborhood of 40° C., accom-

panied by a state of prostration indicating the presence of an acute infective process.

On the other hand, a patient with an uninfected blood collection has little temperature, while the general condition appears to be very fair when compared to the local condition of the injured limb. Still this is not a sign of absolute certitude because the patient may have a high temperature due to some quite different factor than the blood collection and on the other hand the blood collection may become infected at the very start so that the open artery is exposed to a suppurating focus.

The early signs persist when an aneurysmal hæmatoma has become distinctly confirmed, while other symptoms become added. This does not facilitate the diagnosis for the very good reason that they are almost all common to abscess. The tension increases and the limb becomes more painful. Then œdema appears, at first limited to the injured portion of the limb. It is a soft œdema, pitting on pressure, but it does not occur if the collection is deep seated enough not to involve the subcutaneous connective tissue and also if the tension is not too great.

Now, a vast collection of blood filling and distending an entire segment of a limb does not give rise to œdema over the area of the aneurysm. This skin is shiny, tense and presents a reddish blush, while an increased venous network may be seen and are symptoms having the same diagnostic value as œdema.

Below the site of the hæmatoma, over the malleolæ for example, if the case is one of aneurysmal hæmatoma of the lower limb, a non-inflammatory soft œdema exists, whose nature and pathogenesis are quite different from that arising in the neighborhood of the injury. It is an œdema from venous compression, due to the fact that the veins crossing through the cavity of the aneurysm are compressed under pressure by the collection, so that the venous circulation is partially or completely interrupted.

Let me add that the nerve filaments are also irritated and give rise to neuralgic pain and disturbances of nutrition. It is not uncommon to find more or less extensive areas of anaesthesia or hypæsthesia, corresponding to the territory of the nerve involved. The general condition declines, while the temperature hovers below  $38^{\circ}$  C.

A point to which I would particularly draw your attention is *the long duration of the evolution of the process*. An abscess under the same conditions would perforate the integuments in a few days, while a haematoma, even if not absolutely aseptic, remains for weeks in the same state. This fact, gentlemen, is no reason for temporizing when once the diagnosis of aneurysmal haematoma has been made; quite on the contrary, a spontaneous recovery is most exceptional without the occurrence of some serious complication, and it is only by an early surgical interference that such complications can be avoided and which always arise in the evolution of an aneurysmal haematoma when left to itself untreated.

As a matter of fact, infection of the haematoma is very frequent. The manners of infection are numerous. In the first place, the collection occasionally communicates with the entrance or exit openings of the projectile by a small fistulous tract, and these openings are invariably infected, suppurating wounds and are, therefore, the first evident cause of infection of the blood collection. The projectile usually carries the most varied kinds of foreign bodies along with it in its course and deposits them deep in the structures of the limb. Besides the projectile, which itself may remain in the focus of the injury, will be found bits of clothing soiled by contact with the earth, and therefore very septic, bits of stone, earth, etc., all of which are sure factors of infection. Therefore, after a short phase of subacute evolution, an aneurysmal haematoma assumes an acute progress very similar to that of an abscess.

The next complication to be most feared and which is still more serious is gangrene due to cutting off of the circulation. The limb becomes cold and after considerable pain the member becomes anaesthetic. Patches of slough appear, first on the fingers or toes and these extend progressively to the foot or hand and often beyond. A line of demarcation forms, usually within a week and represents the separation between life and death of the part.

The gangrene is first dry and aseptic and if kept properly protected by suitable dressings may remain so. But the areas of slough do not usually maintain this state, so that moist gangrene at length ensues, which darkens the prognosis.

The patient's general condition begins to decline at about this time

and the temperature goes up. The integuments become somewhat jaundiced, indicating a profound intoxication from absorption of the toxic products elaborated in the sloughing structures. If surgical interference is not at once undertaken the outcome will in most cases be fatal. But at present, we do not wait for the evolution of the process to take place, because we treat the case while it is still one of aneurysmal hæmatoma and by so doing we give the patient every chance of avoiding these secondary complications, and if by ill luck we are unable to prevent them each one requires special surgical treatment.

So far I have only referred to aneurysmal hæmatoma resulting from a direct injury to the artery by some type of projectile, but it seems to me that a similar clinical picture is encountered in cases of secondary hemorrhage complicating an abscess due to ulceration of the walls of an artery from the infectious process, or on the other hand, from *a secondary wound of an artery* by some foreign body which has remained in contact with the vessel, the latter being finally opened secondarily by infection. Now, the same clinical picture is present from the time the vessel has perforated as that offered by aneurysmal hæmatoma.

Thus you see how difficult the diagnosis of this surgical affection may be and I know of several instances in which an aneurysmal hæmatoma was mistaken for abscess and only incision of the sac revealed the true state of affairs. When the focus is incised only clots will be extracted at first, but when the pocket has been pretty well cleaned out a jet of blood indicates the true nature of the injury. A mistake in diagnosis is fortunately not serious because both aneurysm and abscess are treated by incision.

Of course it is better to make the diagnosis of aneurysmal hæmatoma before beginning the operation because the principal object of the operation is to ligate the bleeding vessel, so that the incision to be selected is that for the ligature of the given artery only it is made much longer than in ordinary ligature, as much as 15 centimeters or even more if circumstances require.

After having applied some form of constriction above the area of operation in order to procure hæmostasis the focus containing the hæmatoma should be freely incised and after cleaning out the cavity

the two ends of the divided vessel are sought for, at the same time you must carefully inspect the condition of the muscles and other structures which may be included within the sac and to which I have already referred.

The search for the divided ends of the vessel is frequently very long and difficult because the anatomical relations and landmarks have been completely changed by the blood collection which has dissociated the muscles. Also, as I have already pointed out, from the result of maceration both artery and vein are difficult of recognition and sometimes even the veins and nerves have also been injured if the lesion produced by the projectile be extensive so that they cannot be utilized as landmarks in the search for the artery. However, when the nerve trunks exist they are not usually much changed in aspect and consequently are very useful as landmarks. At all events, and no matter how difficult the task, the vascular bundle must be sought for, and if the sheath of the vessel still exists and can be defined it must be slit up freely, after which the distal and proximal ends of the vessel are freed and a haemostat applied to each.

If the ends of the severed artery cannot be found, all that is necessary is to loosen the constricting band so that a jet of blood shall escape from the gaping arterial orifice and then it is an easy matter to catch it in the forceps.

When you have succeeded in clamping both ends of the vessel you will naturally enquire what is next to be done. Suture of the vessel is out of the question, for the macerated and infiltrated vessel walls forbid it, because even a ligature is difficult to apply properly, as it frequently cuts through. Then the large cavity which has been emptied of clotted blood is so greatly disposed to become infected that an aseptic vascular suture can hardly be considered even if the walls of the vessel are in good enough condition to permit of its being done. Therefore, ligature of both ends is the only proper way of dealing with the vessel.

Let me say at once that if it is possible, the artery should be tied at a point which will interfere as little as can be with the collateral branches, but in order to avoid very serious secondary hemorrhage, which might even end fatally, you must be perfectly sure that both the distal and proximal ends of the vessel are *sufficiently*

*resistant to support the ligature.* If not, then those portions of the ends which are doubtful are to be resected so that the ligatures are placed on healthy tissue. I also esteem that in many cases it is wiser not to ligate at all and simply leave the haemostats on the vessel for three days. A considerable experience with vaginal hysterectomy with clamps has demonstrated to my mind the safety of this procedure.

When haemostats are left on they greatly facilitate the drainage of the vast pocket which should be packed with gauze, the clamps being carefully protected by winding strips of gauze around them.

If by chance the aneurysmal haematoma is seated at the root of a limb, a temporary ligature may be placed on the main artery above the haematoma, for example the subclavian in the case of the arm, and then removed at the end of the operation, but it would seem better practice to place a permanent ligature low down on the main vessel, which really means a ligature at the level of the lesion and which greatly facilitates the further steps of the operation.

Bear in mind that in making the ligatures of large arteries that care must be taken to secure a sufficient collateral circulation, that they are successful in portions of a limb possessing many arteries, but are much less so in segments having only one large artery. In the latter case all hope must be placed on the proximal and distal collateral anastomoses and I would say that usually they are quite inadequate for the proper blood supply of an entire limb. In these circumstances gangrene is sure to occur in the territory insufficiently supplied so that amputation must be resorted to and this must be done at a point well above the gangrenous portion of the limb if sloughing of the flaps is to be avoided.

When estimating the possible amount of collateral circulation that may be developed, you must never neglect to take the condition of the vascular system into account, I mean whether or not there is arteriosclerosis, but in the cases of soldiers the men are usually young, with good elastic vessels, which, by dilatation, may compensate for the suppression of one artery. The prognosis will not be so good if sclerosis of the blood-vessels is marked and for this reason, in all doubtful cases, it is well to take the maximum and minimum arterial tension with a Pachon or some other reliable sphygmomanometer, when such an instrument is at hand.

## PULMONARY TUBERCULOSIS AND CONDITIONS SIMULATING IT

A CLINICAL LECTURE GIVEN AT THE HENRY PHIPPS INSTITUTE, APRIL 10, 1918

By H. R. M. LANDIS, M.D.  
Philadelphia

THIS morning I wish to discuss with you the various conditions which may be, and frequently are, mistaken for tuberculosis. There are those who believe that a diagnosis of tuberculosis should be arrived at by exclusion; that is, that a tuberculous infection is to be acknowledged only after you have satisfied yourself that the symptoms and physical signs are due to that cause and no other. On the other hand, there are those who reverse this process of reasoning and hold the opinion that in the event of tuberculosis being suspected you should hold the tubercle bacillus accountable for the manifestations of illness presented by the patient until you definitely prove that some other pathologic process is accountable. Personally I belong to the last-mentioned group. In the first place, pulmonary tuberculosis is by far the most frequent disease of the chest which you will encounter. The mathematical chances, therefore, are always in favor of such a diagnosis in any individual who presents any of the symptoms commonly associated with tuberculosis. In the second place, the failure to keep in mind the seriousness of a failure in diagnosis leads many to mistake tuberculosis for a benign and harmless affection, such as bronchitis.

Ignoring or forgetting these facts is responsible for many cases being overlooked or mistaken for other conditions. The term "protean" has been applied to many other diseases, but of no condition is it so true as tuberculosis. A tuberculous infection of the lungs may ape any disease to which the thoracic viscera are subject, no matter whether it be acute or chronic.

To establish a diagnosis of tuberculosis you are to take into account the history, the location and nature of the pathologic changes, and, finally, the laboratory tests.

1. *The History.*—(a) Does this show that the patient has been

exposed to infection with the tubercle bacillus either by reason of contact with a member of his family or through his associates in his place of employment? (b) Does the history reveal the occurrence of illness in the past which may be considered as reasonably certain to be an evidence of tuberculous infection? There are three symptoms, for instance, the occurrence of which is overwhelmingly in favor of a diagnosis of tuberculosis. First, the history of an haemoptysis. Now, while it is true that blood-spitting can and does occur in a variety of conditions, it is to be looked upon as an evidence of tuberculous infection of the lungs until it can be definitely proved to be otherwise. The occurrence of idiopathic blood-spitting in the temperate zones, therefore, is to be regarded as tuberculous in origin in the vast majority of cases. Secondly, a history of pleurisy with or without an effusion. At the very lowest estimate fully 85 per cent. of all cases of pleurisy are to be regarded as being tuberculous in origin. Insurance companies have for some years recognized the importance of this condition. Their statistics show that of applicants giving a history of having had an attack of pleurisy, four times as many will eventually die of tuberculosis as compared to those without such a history. Thirdly, the occurrence of a fistula in ano is to be regarded as strong presumptive evidence of a latent tuberculous infection of the lungs. It is stated by many that fully 85 per cent. of these fistulae are tuberculous in nature. A history of such a condition is considered by some insurance companies as a cause for rejection.

These three manifestations—haemoptysis, pleurisy, and fistula in ano—are often prodromal symptoms and may antedate definite tuberculous changes in the lungs by years.

A history of lobar pneumonia and typhoid fever is quite common among tuberculous patients. Our experience in the Phipps Institute is as follows: Of 4466 patients, 17.77 per cent. stated that they had had pneumonia; of 5895 patients, 1083, or 18.3 per cent., stated that they have had typhoid fever. Recognizing the fact that tuberculosis is one of the most protean of all diseases and that it frequently manifests itself in an acute form, either under the guise of an attack of lobar pneumonia or of typhoid fever, it is a fair assumption that not a few of the cases just alluded to were of that nature. When it is stated that there have been two or three attacks of pneumonia or typhoid fever, or further questioning shows these attacks to be

atypical, the suspicion is strong that the real trouble has been an acute exacerbation of tuberculosis which has spontaneously subsided.

Finally, the history of the present illness is of the greatest importance. The principal symptoms of tuberculosis are cough with or without expectoration, malaise, digestive disturbances, loss in weight, haemoptysis or blood-streaked sputum, chest pain or distinct pleuritic pain, and slight afternoon fever. All of these symptoms, in the beginning, rarely occur in the same patient. In one malaise will be the predominant feature; in another, the cough; in still another, digestive disturbances will dominate the picture. None of them are pathognomonic of the disease. They become relatively important or unimportant only when taken in consideration with other facts in the history and the physical signs.

2. *Pathological Anatomy*.—No one can hope to achieve any degree of success in any branch of medicine without a definite conception of the pathologic changes involved in the various diseases under examination. Lack of knowledge in this respect is responsible for most of the errors in connection with diseases of the chest. Not only is tuberculosis often overlooked because of failure to take into account the morbid anatomy of the disease, but still more often it is mistaken for other conditions. A tuberculous infection of the lungs as it occurs in adults always first manifests itself at one or the other apex. The initial deposit is a small cluster of tubercles located about one and one-half inches from the apex. From this point the disease has a tendency to spread backward and always downward. Depending on the stage of the invasion—that is, whether the disease is moderately advanced or far advanced—the physical signs will always show the evidence of the greater destruction of lung tissue at the point where the disease first appeared; namely, at the apex. Thus there will be the signs of dense infiltration at the summit of the lung, and as the base is approached the signs will be correspondingly less marked. In advanced cases signs of a cavity at one or the other apex or at both apices is almost the rule.

Keeping in mind the “line of march” of the disease is essential if one is to distinguish between tuberculosis and a number of conditions which, although presenting the same symptoms, have an entirely different pathology.

3. *Laboratory Aids*.—The most important of these aids is the

*examination of the sputum.* This procedure is so simple that it can be done by any one. It is essential, however, to bear in mind certain facts. In the first place, there are several varieties of the streptothrix group which are extremely resistant to acid and are not decolorized by Gabbet's stain or 20 per cent. sulphuric acid. Any organism which resists 30 per cent. nitric acid can be looked upon as certainly being the tubercle bacillus. In the minds of many there still lingers a misapprehension as to the significance of sputum examinations. You not infrequently hear physicians state that the sputum had been examined once and, as tubercle bacilli had not been found, they did not consider that tuberculosis was present. It is to be borne in mind that one negative examination means nothing, no matter what the stage of the disease may be. If you have under consideration a case in the very early stage of the disease, not only one but a dozen sputum examinations may be negative without your being able to say the disease does not exist. Early closed cases often do not show the presence of tubercle bacilli in the sputum. Another mistake in regard to the interpretation of sputum examinations is the failure to heed a number of negative reports when the symptoms and physical signs clearly indicate gross and extensive change in the lungs. In such instances it is far too often taken for granted that tuberculosis is undoubtedly present, in spite of the negative results, and no thought is entertained of other etiologic factors. Again, in cases of the type just mentioned the physician too often takes for granted that the tubercle bacillus is the cause of the trouble and the sputum is not examined at all.

*Tuberculin* tests were at one time resorted to in order to determine the presence or absence of tuberculosis, especially in doubtful cases. For this purpose the subcutaneous injection of tuberculin may be resorted to occasionally. Its routine use is not to be encouraged, and there are some who condemn this method entirely on the ground that it not infrequently arouses into activity a latent and inactive process which may cause serious trouble. The Von Pirquet and Morro tests, which were so universally used a few years ago, are gradually passing into disuse. They are indicative of hypersensitiveness to the tubercle bacillus or its products and, except in children under two years of age, do not distinguish between clinical tuberculosis and healed or quiescent foci.

The *complement-fixation test* has been used to distinguish between cases in which the tuberculous process is active, or about to become so, and those which are healed and which may be disregarded. Its reliability has not yet been determined.

Within the past few years the *X-rays* have been lauded as the only sure and reliable method of determining the presence of tuberculosis and also of distinguishing the disease from other affections of the lungs which may simulate it. If one is to believe what the ultra-enthusiastic röntgenologists claim, it is a waste of time to spend our efforts in trying to find out the cause of the trouble by the usual methods of physical diagnosis. Baetjer put the matter very succinctly when he stated that the only point at issue is an etiologic diagnosis in the early stages of the disease. As he has pointed out, this is not possible by means of the X-rays, as a number of other conditions give rise to changes in the lungs and bronchi which are not to be distinguished from tuberculosis. By the time the X-ray picture is definitely that of tuberculosis the clinical evidence is so clearly apparent that the need of an X-ray examination is superfluous. My own experience is that in the vast majority of cases the time-honored clinical methods are efficient: in a relatively few the X-rays will detect changes which have escaped the clinician; and, on the other hand, a few will escape the röntgenologist which are detected by the clinician. Within the past few months I have knowledge of three cases in which an experienced röntgenologist reported negatively as to the presence of tuberculosis when all three of the cases showed the presence of tubercle bacilli in the sputum.

*Acute Bronchitis*.—Cough is the most frequent, and often in the beginning the only, symptom of an early tuberculosis. Attributing a cough of this nature to an attack of acute bronchitis is one of the most frequent errors made. This is largely due to the slight and indefinite physical signs which are often elicited in the early stages of tuberculosis. In the absence of anything definite the cough is assumed to be negligible and probably an evidence of acute bronchitis or trachitis. A safe rule to follow in such cases is to consider a cough which has lasted for six weeks or longer, and for which there is no adequate explanation, as being strong presumptive evidence of a beginning tuberculosis. The longer such a cough has been present the more suspicious one should be that it is not due to a simple

benign bronchitis. The latter, in the vast majority of cases, has a duration of three or four weeks; rarely does an acute bronchitis become subacute and continue indefinitely. The young man I am about to show you has been a victim of such a mistake. Six months ago he developed a cough, for which he consulted a physician after it had been present six weeks. He was told that he had bronchitis, and was treated, until two weeks ago, for this condition. During this time the cough has steadily become worse and the sputum more and more profuse, in spite of the use of various cough remedies. On several occasions the sputum was blood streaked. In addition, he gradually lost twelve pounds in weight and suffered from an increasing malaise. Recently he developed slight huskiness of the voice. In spite of assurances that the condition was nothing more than a bronchitis, he himself became apprehensive that all was not well, and on his own suggestion his sputum was examined and found to contain many tubercle bacilli. Examination of his chest shows distinct diminution of motion down to the fourth rib on the left side, and over the greater part of the left upper lobe the breath sounds are suppressed, and after cough showers of fine crackling râles are heard. The disease has now progressed to a point where recovery is doubtful; the best that can be hoped for is an arrest of the process and a limited sphere of activity for him the rest of his life. You may think this is an exaggerated instance. So it is, but none the less it is one that is encountered with considerable frequency.

You will note and bear in mind the two cardinal mistakes in this case: First, that of regarding as simple bronchitis a cough which had persisted for months; and, second, the failure to examine the sputum at the very onset of the trouble.

*Chronic Bronchitis*—This condition is not often primary. In the great majority of instances it is secondary to some other condition. It is commonly encountered in those past the middle period of life who are asthmatic or emphysematous. Among those who suffer from emphysema a chronic winter cough is common. During the summer months the cough is very slight or it may disappear entirely. Overlooking a tuberculous infection and mistaking it for chronic bronchitis usually occurs in one of two ways. In the first place, it is often assumed that a cough which has persisted for months must be due to a chronic bronchial condition. In the second place, when such

a condition actually is present, there is a failure to realize that a tuberculous infection is also present. In this latter group the emphysema often marks the physical signs of the tuberculous process. Furthermore, the constitutional signs in these cases are often slight and do not attract the attention of either the patient or the physician. Again, there are not a few cases of tuberculosis which pursue a latent course and in which there are considerable fibroid changes in the lungs. In such cases there may be scattered râles throughout both lungs. This fact, taken in conjunction with a chronic cough and little or no failure in the general health, leads the unwary to overlook the possibility of the serious lesion and look upon the process as a chronic bronchitis. While in the majority of these cases the experienced physical diagnostician is not deceived, an error can be avoided by every one if the sputum is examined. Indeed, it should be a fixed rule to do this in all cases of chronic cough, no matter how slight the chances are that a tuberculous infection may be present.

*Bronchiectasis.*—An extremely common error is the mistaking of bronchiectasis for tuberculosis. Dilatation of the bronchi occurs in practically all instances in which extensive fibrosis of the lungs has taken place. It occurs in its most characteristic form in the massive type of fibrosis where an entire lobe or even one entire lung has undergone fibroid changes. The etiology is varied. Any condition which causes inflammatory changes in the pulmonary tissue may gradually produce a fibrosis and, later, dilatation of the bronchi. Tuberculosis itself is a frequent cause of both fibroid changes in the lungs and dilatation of the bronchi, but in these cases the bronchiectasis is not usually so marked as in the cases of non-tuberculous fibrosis. Dilatation of the bronchi is more commonly encountered at the base of the lungs than at the apices. It is to be borne in mind, however, that the condition does occur at the apices more often than is usually thought.

The usual text-book description of bronchiectasis is that the condition is characterized by the expectoration of large quantities of sputum which often is of an extremely foul odor. This emptying of the bronchi takes place two or three times a day, and during the rest of the time there may be little or no expectoration whatever. Out of a very considerable number of cases of bronchiectasis which I have seen the expectoration of very large quantities of very foul sputum is

the exception rather than the rule. In the majority of cases the sputum, while profuse, is not always foul, nor does it come up in very large quantities at infrequent intervals.

The symptoms of bronchiectasis are cough, expectoration, blood-streaked sputum, or small haemoptyses. The cough may be paroxysmal and attended with profuse expectoration. Owing to the associated fibrosis there may be some shortness of breath. Constitutional symptoms are usually absent in spite of the foul secretions in the bronchi.

In the case I now show you the most noticeable feature on inspection and the one that attracts our attention at once is the extreme clubbing of the fingers and the curving of the nails over the ends of the fingers, the so-called "parrot-beaked" finger-nails. Whenever you see clubbing of this type you can be almost positive that you have to deal with extensive fibrosis of the lungs and an associated bronchiectasis. Extreme clubbing is sometimes seen in cases of empyema; it is also a constant feature in congenital heart-disease, but in this condition the ends of the fingers are also cyanosed. While clubbing of the fingers is very frequently seen in tuberculosis, it is rarely of the extreme type encountered in cases of non-tuberculous fibrosis of the lungs.

Next to the extreme clubbing of the fingers, it is to be borne in mind that the abnormal physical signs, as in the present case, are commonly confined to the base of one or the other lung. This fact is almost proof positive that the condition is not tuberculous. The man I now have before you has been under observation for nearly twelve years; his sputum is negative for tubercle bacilli, and the physical signs consist of limitation of motion at the right base, distant breath sounds, mixed râles, and the signs of a cavity near the angle of the scapula. The latter condition is commonly present in these cases and is due to dilatation of the bronchi at the root of the lung. At the present time I have two such cases under my care at the White Haven Sanatorium, both being sent in under the belief that they had tuberculosis.

One word more as to this condition: I have already referred to the dilatation of the bronchi being limited to one or the other apex of the lung. In such cases the symptoms and physical signs (especially that of cavity) are identical with those encountered in tuberculosis. Clubbing of the fingers in this type is absent, probably because the drainage is good and the secretions do not stagnate in the bronchi.

Examination of the sputum is the only certain means of distinguishing apical bronchiectasis from tuberculosis. If the sputum is repeatedly negative for tubercle bacilli a diagnosis of fibrosis of the lung and dilatation of the bronchi is permissible.

*Pneumoconiosis.*—Closely allied to the above condition is pneumoconiosis. Although this condition is described in all the text-books, it rarely is referred to in our daily work. A diagnosis of pneumoconiosis is hardly ever used in either the morbidity or mortality returns. The secondary changes which are caused by the inhalation of inorganic dust are the ones usually referred to, such as chronic bronchitis, emphysema, asthma, etc. Now that more and more interest is being taken in industrial diseases the term pneumoconiosis as indicative of a distinct pathologic process will probably be more and more used.

The condition arises under the following circumstances: If a worker is exposed to inorganic dust (notably that containing much silicia) for a period of years the fine dust particles are carried into the respiratory tract and gradually set up a chronic inflammatory condition. This eventually leads to extensive bilateral fibroid changes constituting a fibrosis of the lungs. It is important to know that after the condition of pneumoconiosis becomes well established the changes are more noticeable in the right lung than the left, and that, furthermore, the changes are often more pronounced about the hilus and the apices. To one who is unfamiliar with the fact, both physical signs and an increase in the shadows of the X-ray plate, more marked at the apices, might be interpreted as being due to tuberculosis. When the process has reached an advanced stage both lungs are markedly scarred and the physical signs are indicative of gross pathologic changes. Pneumoconiosis may lead to extensive fibroid changes which later has added to the picture a tuberculous infection. The mistake is often made of calling pneumoconiosis tuberculosis because of the similarity of symptoms and the physical signs. It is not possible to differentiate the two except by examination of the sputum. The presence or absence of tubercle bacilli will be the determining point.

This condition serves to emphasize the importance of knowing the occupation of the patient. Whenever it is stated that the occupation is that of a potter, a coal miner, a metal grinder, or, in fact,

any employment in which inorganic dust is a factor, the diagnosis of pneumoconiosis should at once suggest itself.

*Malignant disease* may involve the lungs primarily or secondarily, and may be either carcinomatous or sarcomatous in nature. Among other types of malignant disease which affect the lungs or pleura may be mentioned endothelioma, and secondarily hypernephroma, and malignant deciduoma.

Primary malignant growths of the lung are rare and the diagnosis is not easy. It is usually made by exclusion. These cases have a cough, some expectoration, blood-tinged sputum, they emaciate and lose strength, and usually have an irregular type of fever. Occasionally the sputum is markedly hemorrhagic and presents the appearance of currant jelly. When present, sputum of this kind is extremely suggestive of malignant disease. Dyspnoea is often a marked feature and is out of all proportion to the amount of damage in the lungs as demonstrated by the physical signs. In a case seen here in the Institute several years ago extreme dyspnoea was about the only symptom present. The physical signs consisted of nothing more than the presence of a few râles scattered throughout both lungs. At the autopsy not more than four or five nodules, about the size of a walnut, were found in each lung. The primary growth in this case was a small unrecognized scirrhous cancer in the left breast.

Primary malignant tumors occur by preference about the root of the lung or at the bases—a valuable point in the differential diagnosis. They may, however, be limited entirely to an upper lobe. The sputum should be repeatedly examined in order to definitely rule out the presence of tubercle bacilli. Recently several excellent röntgenologic studies have been made of malignant disease of the lungs and these have shown that the X-ray picture is quite characteristic of the condition. All doubtful cases should be subjected to such an examination.

Secondary or metastatic malignant disease of the lungs is not uncommon and, as a rule, is easily recognized. Any individual who has had a malignant growth removed or who is known to have such a growth should be suspected of having a metastasis of the lungs, if respiratory symptoms develop.

Malignant disease of the lungs is overlooked far too frequently, and in every instance the error is to be attributed either to the fact

that the sputum is not examined or that the warning of repeated negative examinations is not heeded. Given a patient who is obviously going down hill, who has marked respiratory symptoms, and in whom the physical signs are indicative of gross pathologic changes in the lungs, some other explanation of the trouble must be sought for if the sputum, after at least half a dozen examinations, fails to show the presence of tubercle bacilli. That the mistake is a common one is attested to by the fact that institutions devoted to the care of tuberculosis patients are constantly having sent to them individuals who really have malignant disease. Thus in one institution a recent report shows that out of 198 autopsies on supposedly tuberculous individuals five proved to be due to malignant tumors of the lungs.

*Lobar Pneumonia.*—It is not sufficiently recognized that, while tuberculosis, as a rule, is characterized by an insidious onset, it very often begins abruptly and simulates one of the acute infectious diseases. This is particularly true of lobar pneumonia. Not infrequently tuberculosis will first manifest itself with a chill or chilly sensations, pain in the side, cough, bloody expectoration, and a high continuous type of fever. Such an onset is identical with that of pneumonia, and for a week or ten days the two conditions are not to be differentiated clinically. Furthermore, there will be the signs of partial or complete consolidation. It is worth remembering, however, that it is the apex or upper lobe which is always the site of the abnormal signs if the condition is tuberculous; if due to croupous pneumonia, the apex may be involved, but the base of one or the other lung is the common site. In such cases no suspicion is entertained that the condition is other than a croupous pneumonia until ten days or two weeks have elapsed, when, if the condition is tuberculous, the temperature begins to fluctuate, crisis does not occur, sweating is apt to take place, and the sputum becomes purulent. Examination of the sputum will now show tubercle bacilli to be abundantly present. It may take several weeks to determine the question, however, if the pneumonia occurs in an individual who already has tuberculosis, because in these cases a rapid spread of the tuberculosis may be attended with symptoms similar to those just mentioned.

The process is to be suspected as being tuberculous from the very beginning in all cases of apical involvement if there is a family history of tuberculosis or if the individual gives a history of having

been in ill-health for some time, or if he has had a slight hacking cough for several months. It is frequently stated by patients that they have never been well since they had an attack of pneumonia some months previously, or the physician states that the tuberculosis developed as a result of the pneumonia. For some reason, hard to explain, it has long been known that when a true lobar pneumonia does occur in a definitely tuberculous patient it has little or no effect on the tuberculosis. Therefore one can usually be pretty certain that when it is said pneumonia caused the tuberculosis such is not the case, but the trouble was tuberculous from the very beginning.

*Syphilis.*—There are many who refuse to accept a diagnosis of syphilis of the lung or, at any rate, believe that it is encountered only very rarely. Since the discovery of the *Spirocheta pallida* and the Wassermann reaction, especially the latter, more and more reports are appearing on the subject of pulmonary syphilis. Personally I am of the belief that the condition occurs with greater frequency than is at present believed.

The diagnosis is always a matter of excluding all conditions which may account for the symptoms presented except syphilis. Without going into the subject exhaustively, I will relate the facts in one case and show you another. These cases are among some eight or nine observed here at the Institute of what we believe to be syphilis of the lung. Several years ago we had under observation a married woman of thirty-three years of age who applied for treatment because of a cough of some months' duration, yellowish expectoration which was blood tinged on several occasions, loss in weight, and slight, irregular fever. Abnormal physical signs were present at the right apex. The diagnosis seemed to be evident, although tubercle bacilli were not found. This was accounted for because of the slight amount of pulmonary involvement, the assumption being that the lesion was a closed one. She attended the dispensary regularly, but at the end of eight months her condition was unchanged. Finally, about this time she complained of severe nocturnal headache. We then noted, what we had previously disregarded, that her history stated that she had had three miscarriages—a fact that should have aroused our suspicions from the beginning. A Wassermann test proved to be strongly positive. Two doses of salvarsan, followed by mercury and

iodide of potassium, led to a disappearance of the fever, a gain in weight, and a rapid subsidence of the cough.

The man I now show you is, as you see, apparently in robust health. When first seen, two years ago, he had not been able to work for four or five months. He had lost a great deal of weight, had a slight afternoon fever, a cough and expectoration, which was blood tinged at times. In addition, he felt very weak. Examination of his chest showed that there was some diminution of expansion, slight impairment of the percussion note, feeble breath sounds, and fine râles at the right apex. Owing to the fact that he gave a clear history of a luetic infection eighteen years previously, a Wassermann test was made and found to be strongly positive. His sputum was negative for tubercle bacilli. We therefore considered him to be suffering from syphilis and not tuberculosis. Salvarsan, followed by mixed treatment, caused a disappearance of the symptoms and a gain in weight of about forty pounds. He has been back at work and perfectly well for about eighteen months. There can be no doubt that there are not a few individuals presenting symptoms such as these two patients have who are treated for tuberculosis when in reality the true nature of the trouble is a latent syphilis.

Syphilis is to be suspected in a woman who gives a history of several miscarriages, in a man if one or both testicles are hard and indurated, or in either in case of enlargement or tenderness of the sternal ends of the clavicles or ribs. These are among the commoner of the syphilitic stigmata. It is to be borne in mind that both syphilis and tuberculosis may exist in the same individual.

*Mycotic Infections of the Lungs.*—Infection of the lungs with some one of the yeasts or moulds may occur. Although the total number of such infections is not great, they should always be kept in mind in cases which present every evidence of tuberculosis but in which tubercle bacilli are persistently absent from the sputum. In such an event the sputum should be studied from the standpoint of other possible infecting organisms. Every now and then one is rewarded by the finding of one of these unusual infections. In the order of their frequency the following conditions may be encountered: streptothricosis, actinomycosis, blastomycosis, coccidioidal granuloma or California disease, aspergillosis, and sporotrichosis. All of these conditions are characterized by cough, mucopurulent and, at times,

blood-tinged sputum or small haemoptyses, pain in the chest, loss of weight, and fever. While they may invade any portion of the lung, the infiltrated area is frequently limited to one apex, or, when first seen, a large portion of a lung may be uniformly invaded. Unless the lesion is localized at the base of the lung, there is nothing about these cases to indicate that they are other than tuberculous in character. The key to the situation is the examination of the sputum. If this is repeatedly negative for tubercle bacilli, another source of the trouble is to be sought for.

It may be worth while mentioning that several of these mycotic infections have a curiously restricted geographical distribution. The great majority of cases of systemic blastomycosis which have been reported in this country come from the immediate vicinity of Chicago. A single instance of this disease has been observed here in the Institute. Coccidioidal granuloma or California disease is restricted, apparently, to a single district—the San Joaquin Valley, in California. Of the forty cases so far recorded, all but one or two have occurred in this area. Sporotrichosis, which usually manifests itself as a skin lesion, although the lungs are occasionally involved, has been observed in this country almost exclusively in the region comprising the Mississippi River basin; of 73 cases thus far reported, 68 have come from this locality. The majority of the cases of pulmonary aspergillosis have been observed in France. It has been noted among those who handle grain and in men engaged in the forcible feeding (mouth to beak) of pigeons. The others have a very wide distribution, especially the streptothrix group, which is by far the commonest of all.

*Pulmonary Distomatosis.*—This condition is also known as lung-fluke disease, parasitic haemoptysis, and endemic haemoptysis. It is deserving of mention, for, although the United States are not included in its geographical distribution, it may be encountered at any time in those who have resided in the Far East. The disease occurs endemically in Japan, China, Korea, and the Island of Formosa. The principal symptoms are chest pain, cough and blood-streaked sputum, or a frank haemoptysis. The diagnosis rests on the absence of tubercle bacilli and the presence of the eggs of the worm in the sputum. The eggs are golden yellow in color and larger than the eggs of other parasites common to man. They may be seen with the unaided eye as brownish specks if a small portion of the sputum con-

taining them is pressed between a cover-slip and slide and held up to the light. As both tuberculosis and distomatosis may occur in the same individual, both conditions should be kept in mind in those who have lived in the endemic zone.

*Mitral Stenosis.*—At first sight it would not seem likely that a cardiac lesion would give rise to mistakes. In all institutions devoted to the care of tuberculous patients, however, it is not an uncommon occurrence to have sent to them cases of chronic heart-disease, particularly mitral stenosis, in the belief that tuberculosis is present. This is to be ascribed to the fact that such patients often have a cough, blood-tinged expectoration, shortness of breath, and often the characteristic pre-systolic murmur and thrill are not present. In addition, the sputum is either not examined or the significance of several negative examinations is not heeded. In this frozen section which you now see you will note that the lungs are unusually healthy in appearance and that the heart is greatly enlarged. This specimen was obtained from the anatomical department of the University of Pennsylvania. The cause of death, in this case, was given as pulmonary tuberculosis.

*Thoracic Aneurism.*—The signs of aneurism of the arch of the aorta, particularly the transverse and descending portions, are often obscure and pass unrecognized. Very often there will occur in such cases considerable chest pain, cough, and bloody expectoration. In addition, patients suffering from aneurism are often somewhat emaciated and have a slight amount of fever. Furthermore, as the tumor may extend toward the apex or, because of compression on a main bronchus, cause a feeble respiratory murmur on one side, the signs, taken in association with the symptoms, lead to an erroneous diagnosis of tuberculosis. It is well to remember, however, that the two conditions are not uncommonly associated. So far as aneurism is concerned, the easiest way of determining its presence is by means of an X-ray examination. Patients complaining of vague but persistent substernal pain should always be subjected to such an examination.

In briefly considering these various conditions which bear, in some instances, a close resemblance to pulmonary tuberculosis, and in others only a superficial likeness, it has been my desire to point out the fact that in nearly every instance a mistake can be avoided if certain well-established rules are kept in mind. First, the examination of the sputum. This should never be neglected in any case in which tuber-

culosis is suspected. In the very early cases it is, as a rule, negative, but in those cases in which the presence of râles at the apex indicates the presence of moisture the sputum should be examined a sufficient number of times to make it reasonably certain that another cause of the trouble is to be sought for.

Where the signs and symptoms indicate the presence of a well-marked tuberculous lesion it should never be taken for granted that tuberculosis is, of necessity, the true diagnosis. It is because of neglect in such cases that many instances of bronchiectasis, pneumoconiosis, mycotic infections, malignant disease, and even cardiorenal disease are mistaken for tuberculosis. A single negative examination in a case with extensive pulmonary damage demands that the examination be repeated until tubercle bacilli are actually found or until a sufficient number of negative examinations make it clear that some other cause of the trouble must be thought of. Secondly, it is essential to keep in mind the morbid anatomy of tuberculosis. A lesion occurring in one of the upper lobes may or may not be tuberculous; one limited to the base of the lung is almost certainly not tuberculous. Finally, remember that, while there is no symptom which is pathognomonic of tuberculosis, the occurrence of an haemoptysis or an attack of pleurisy or the presence of a fistula in ano is the strongest kind of evidence in favor of the trouble being tuberculous.

## CLINIC OF DRs. JULIUS H. HESS AND A. LEVINSON

DEPARTMENT OF PEDIATRICS, UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE

### SKIN REACTIONS IN CHILDREN

In the small series of cases which we show this morning we will endeavor to demonstrate the practical value of the Schick reaction for the diagnosis of the presence or lack of immunity to diphtheria toxin, the luetin reaction in syphilis and some of the food reactions.

#### THE SCHICK TEST

**CASE I.**—This is one of a series of cases in which the Schick test was applied in the wards of the Cook County Hospital during the present epidemic of diphtheria in the Children's Building. You will notice on the arm a sharply defined area of redness, slight infiltration of the same being palpable (Fig. 2, frontispiece). This is a typical papule which is the result of injection of 1/50 m. l. d. of diphtheria toxin performed seventy-two hours ago. We believe from the typical reaction noted in this case that this child is susceptible to diphtheria toxin and should, therefore, be protected by the early injection of a suitable dose of antitoxin.

**CASE II.**—This child taken from the same ward as Case I and subjected to the same treatment shows no reaction and may be described as a negative case. We may, therefore, conclude that this child is sufficiently protected with antibodies to remain immune to the disease, without an injection of antitoxin.

**CASE III.**—This is the nurse who has been in attendance in this ward and who has been tested similarly to Cases I and II. She gives a positive reaction similar in character to that seen in Case I. For her own protection she should either receive a dose of antitoxin or if a longer protection is desired, both antitoxin and toxin-antitoxin, as suggested by Park and Zingher.<sup>1</sup>

We have before us three individuals all exposed to the same infection, all of them showing negative throat cultures, two of whom are susceptible and the third of whom is immune to the diphtheria toxin.

This test, therefore, gives us valuable information on which to base prophylactic treatment.

Schick<sup>2</sup> discovered that diphtheria toxin, when injected intracutaneously in individuals not sufficiently protected by antibodies, will produce a specific skin reaction, which in turn may be influenced by injection of diphtheria antitoxin. Upon this principle he based a test, which enables one to tell whether or not a person has sufficient diphtheria antitoxin in his system to confer upon him immunity from the disease.

The technic of the test consists of injecting 1/50 of a minimal lethal dose for a guinea pig weighing 250 grams. The injection is made intracutaneously. The cultivation of the organisms for their toxins, and the standardization of the toxin will not be detailed at this time, as the undiluted diphtheria toxin can be obtained in the open market either in vials of 1 or 2 mils or in single outfitts. The bulk toxin should be one that has been ripened for at least one year, and it should at all times be kept away from light and kept cold (45 degrees F. or less). The toxin is diluted with sterile salt solution. This solution is injected into the epidermis with a fine, sharp, but short bevelled needle, preferably one of platinum-iridium, so that a definite wheal-like elevation with a distinct marking of the opening of the sweat glands will result at the point of injection. Fortunately the practical application of the test has been simplified by marketing of a simple carton containing a complete outfit of one unit with the necessary sterile solution, and tubes for proper dilution. Such an outfit will, if necessary, supply sufficient toxin for the vaccination of fifty cases.

A control test should be made by injecting into the other arm in the same dilution and from the same stock solution diphtheria toxin which has been either heated to 75 degrees C. for five minutes to destroy the soluble toxin, or has been over-neutralized by the addition of the antitoxin. This mixture is prepared by adding two units of antitoxin to one unit of toxin.

A positive reaction represents the action of an irritant toxin on unprotected cells of the body. If the individual upon whom the test is being made is immune to diphtheria, that is if his body fluids contain a sufficient amount of antitoxin, the toxin is neutralized im-

mediately after its injection and is thus unable to evolve any action upon the cells of the body. Schick found that the amount of anti-toxin necessary to suppress a diphtheria toxin reaction is 0.03 of a unit per mil of blood. When, however, antitoxin is either absent or present in the body in lower concentration than stated above, then injection of toxin is followed by inflammation and cellular infiltration at the place of injection.

A trace of redness appears slowly at the site of injection in from twelve to twenty-four hours, and usually a distinct reaction in the course of twenty-four to forty-eight hours. At this time the positive reaction appears as an area of redness with a definitely sharp outline, but only slightly elevated, the infiltration not being marked. The size of the reaction is usually from 1 to 2.5 cm. in diameter. In individuals whose skin is more susceptible the reaction is more marked, infiltration being more evident, at times giving rise to a vesicle. The more toxin injected, the more violent the reaction, as shown by Cowie.<sup>3</sup> The reaction reaches its height on the third or fourth day and gradually disappears, leaving a definitely circumscribed scaling area of brownish pigmentation, which persists for three to six weeks.

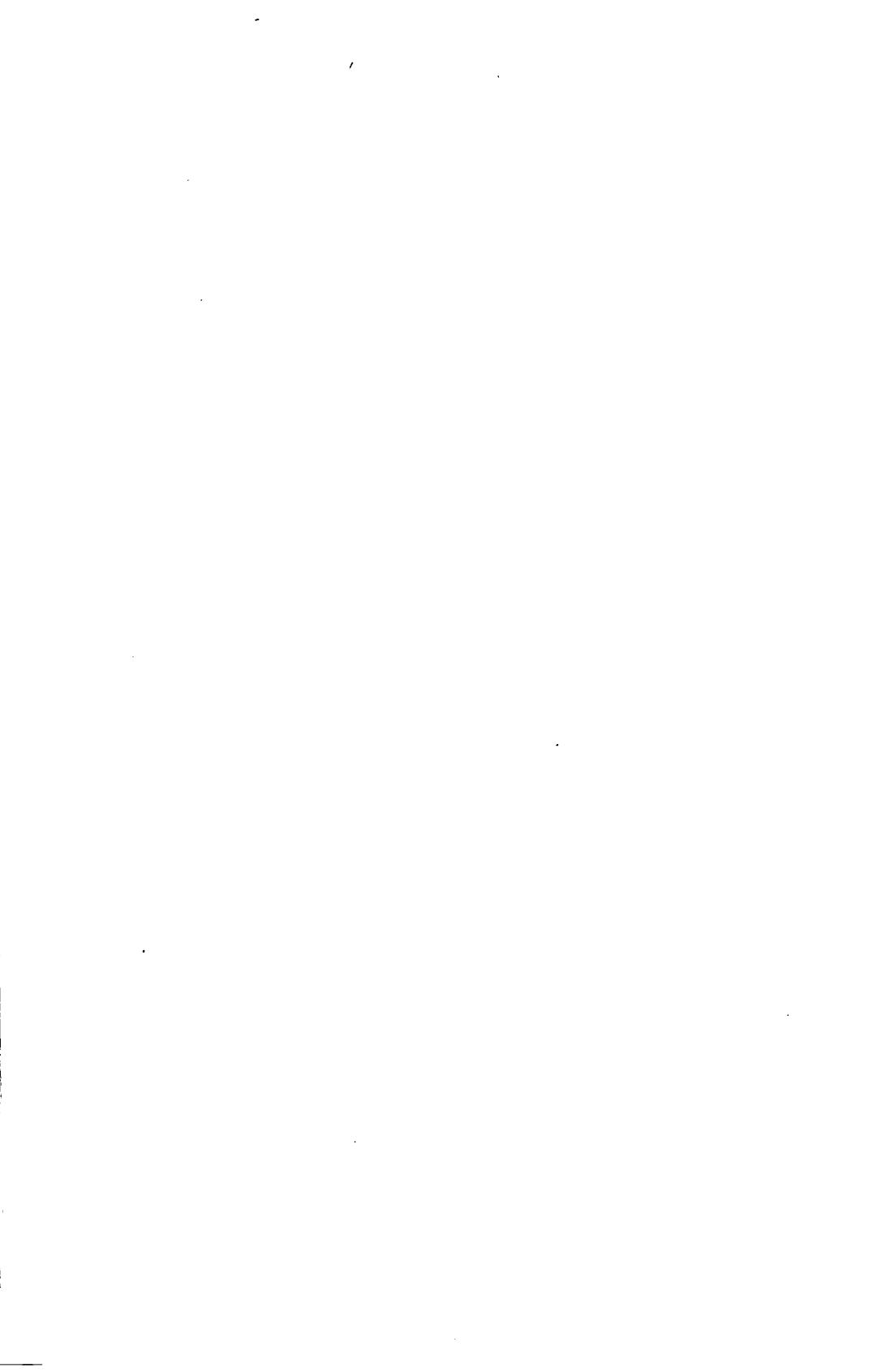
When the reaction is absent or negative no change is noticeable in the skin at the place of the injection of toxin, since the toxin has been neutralized and thus failed to act as an irritant.

Of considerable importance in interpreting the results of the test are the so-called pseudo-reactions which occur mostly in persons suffering from or convalescing from scarlet fever or measles. These pseudo-reactions may be due either to trauma, to hypersensitiveness of the skin or to the various constituents of the broth in which the bacilli were cultivated during the production of toxin, or, on the other hand, to diphtherin which is a protein substance of the autolyzed diphtheria bacilli. The first group of factors may be eliminated by refinement of the technic and by the use of potent stock toxins. The pseudo-reaction due to diphtherin is an anaphylactic response of the tissue cells to the protein substance of the autolyzed bacilli, and like other anaphylactic skin phenomena, the reaction is of an urticarial nature, appears early, within six to eighteen hours, reaches its height in thirty-six to forty-eight hours, and disappears on the third or fourth day, leaving no pigmentation or only a poorly defined, small, brownish

FIG. 3.



Diphtheria toxin reaction (Schick Test). Positive reaction 48 hours after inoculation.



spot. At its height the pseudo-reaction shows varying degrees of infiltration, and appears as a small central area of dusky redness, with a secondary areola, which gradually shades off into the surrounding skin.

To differentiate this pseudo-reaction from a true reaction a control injection is made, as previously described. The destruction of soluble toxin before injection removes the toxin factor of the reaction and if, therefore, after such an injection a skin reaction still appears, we know that it is not due to the toxin, which has been removed from the fluid before the injection, but that it must be due either to diphtherin or to some other factors, as previously mentioned. Thus, if the original test was a pseudo-reaction, the control will show a similar area of redness and infiltration and both reactions will run the same clinical course, namely, disappear in three to four days and leave little, if any, pigmentation behind.

*Combined Reactions.*—Occasionally one sees a combined reaction which represents both a positive and a pseudo-reaction. The central area of redness is larger and better defined. The amount of infiltration is more marked also. To interpret such a reaction properly we should obtain the evidence of a true reaction, a definite area of scaling brownish pigmentation after the pseudo-element has disappeared in the test. In addition, we should obtain similar, though weaker reaction in the control test made with heated or overneutralized toxin. The control, will, of course, represent only the pseudo-reaction. These pseudo-reactions are rarely seen in the young, but quite frequently in the adult. Unless one is able carefully to differentiate them from the positive reactions it will be safer in practice to consider all reactions as positive, and treat them as such on exposure to diphtheria. Recently it has been found that when the dose of toxin is contained in 0.1 mil of fluid as originally advocated by Schick, fewer pseudo-reactions occur than when the toxin is contained in 0.2 mil of fluid.

*Recording the Test.*—For the purpose of recording the tests we may designate the reactions by the following signs, which represent varying degrees of intensity. This is especially important to the active immunization with the mixture of diphtheria toxin and anti-toxin when we wish to know the degree of the original or control test

before the immunizing injections, and of tests made at intervals after the injections.

++ strongly positive. Marked redness and considerable infiltration, occasionally superficial vesiculation.

± positive. Redness and little or no local infiltration.

± moderately positive. Varying degrees of redness and no local infiltration.

± faintly positive. Slight redness and no local infiltration.

— negative. No redness and no local infiltration.

Levinson and Blatt<sup>11</sup> call a reaction positive when there is an erythema and induration of at least  $0.5 \times 0.2$  cm. and does not disappear in twenty-four hours.

*Immunity to Diphtheria as Shown by Schick Test.*—Zingher<sup>4</sup> tested 2700 children of various ages from two to sixteen years, and found that 580 gave a positive reaction, with the highest percentage of 32.2 between two and four years of age, and with a gradually decreasing percentage to 16.4 between fourteen and sixteen years of age. This group were normal children who resided in three different orphan asylums. In his cases he found very few pseudo-reactions, but about 10 per cent. of combined reactions.

Park, Zingher and Serota<sup>5</sup> performed Schick reactions on 700 cases of scarlet fever patients and found that 43 per cent. gave a positive reaction.

In another group of 1200 children suffering with scarlet fever Zingher found a much larger proportion. In these latter cases he found 65 per cent. of positive reactions in those between two and six years of age. He believes that this possibly is due to the fact that the higher temperature of scarlet fever with the associated increased metabolism of the body causes a breaking down of the trace of natural antitoxin, which is just sufficient to give a negative reaction in some of the healthy children; possibly also, those who are susceptible to scarlet fever are more apt to be susceptible to diphtheria, that is to give a positive Schick reaction.

Veeder<sup>6</sup> reports 7 per cent. of positive reactions on 291 new-born infants. In 42 cases less than one year old he found 43 per cent. positive reactions. Between two and five years 63 positive. Moffet and Conrad<sup>7</sup> also report a higher percentage of positives than those of Zingher.

Cowie (*loc. cit.*) has performed Schick tests on eighty-one persons taken at random, ages varying from two days to adult life. Fifty reacted positively and thirty-one negatively. The age incidence of reaction of the eighty-one patients was, as follows:

0-5 months 23 per cent. positive.  
5-12 months 83 per cent. positive.  
1-5 years 87 per cent. positive.  
6-12 years 52 per cent. positive.  
Adults 77 per cent. positive.

In twenty-three infants, ages varying from two days to ten months, on whom Schick tests had been made by Cowie, there were 10 positive and 13 negative reactions, which is 56.5 per cent. negative.

Moffet and Conrad (*loc. cit.*), Moody<sup>8</sup> and Cowie (*loc. cit.*) found that Schick reaction is more often positive in winter months and less frequently in summer months, the average figures giving 31 per cent. positive in summer and 58 positive in winter.

Kolmer<sup>9</sup> sums up the practical value of the Schick test, as follows: The intracutaneous toxin test of Schick for antitoxin immunity in diphtheria may be stated to have definitely established itself as a safe, easily applied and reliable practical test for detecting those persons who have sufficient natural diphtheria antitoxin in their body fluids to protect them against this infection and also as a measure of antitoxin production after active immunization with toxin-antitoxin mixtures, the latter being the original purpose of Schick in working out the technic of this test to replace the more expensive, time consuming and laborious procedure of determining the antitoxin content of the serum by means of injecting mixtures of the serum and a toxin into guinea-pigs.

Of great interest is the question of the reaction of Schick simultaneously with some other toxin tests. Schick and Maygar<sup>10</sup> tested 315 individuals with both tuberculin and diphtheria toxin at the same time and obtained the same reaction (both positive or both negative) in 163 cases, and different reactions in 153, almost 50 per cent.

Levinson and Blatt<sup>11</sup> have tested fifty-five children that were afflicted with diseases other than diphtheria and tuberculosis and found thirty-four, or 61.8 per cent., showing negative Schick reaction.

They also tested thirty-three children who received the Von Pirquet test, and thirty-eight children who received tuberculin treatment simultaneously with the Schick, and found that the reaction bore no relation to the Von Pirquet test or to the tuberculin treatment.

The Schick test, which is based on injection of diphtheria toxin, the product of the bacillus in culture medium, and the injection of diphtherin which represents the killed bacteria, must not be confused.

Regarding the practical value of diphtherin reaction Kolmer (*loc. cit.*) comes to the following conclusions after extensive study of the subject: "While the diphtherin test indicates hypersensitivity to the protein of diphtheria bacillus, it has probably no value as an index of immunity and is of practical interest mainly from the viewpoint that the anaphylactic reaction may be mistaken for a positive Schick reaction."

Diphtheria carriers are immune to diphtheria toxin and usually have comparatively large amounts of antitoxin in circulating blood.

The test is of greatest value in institutions, schools and families during epidemics to select those individuals who have been or will be exposed to diphtheria infection and who are not immune to diphtheria toxin. There are two advantages derived from positive knowledge of the state of susceptibility to diphtheria toxin: (1) Saving of diphtheria antitoxin by using it only in individuals in whom the Schick test is positive, and (2) avoiding dangers of anaphylaxis to horse serum and other disagreeable sequelæ in individuals who by negative Schick test show that they do not need the antitoxin.

*Conclusions.*—(1) A positive reaction being a sign of lack of immunity is an indication for active immunization by injection of antitoxin or toxin-antitoxin into individuals who have been or are going to be exposed to diphtheria infection. Thus the test is of utmost value in preventive medicine.

(2) The greatest possibilities for its use are found in institutions for housing of children, and in the presence of epidemics, and where other children of the same family have been exposed. It can also be used to advantage in testing school children with the assurance that a negative reaction in all probability means at least temporary protection.

## THE LUETIN TEST

The case, which we show you this morning, is that of a girl, age thirteen years, who presents certain definite lesions which are indicative of syphilis: typical Hutchinson's teeth, interstitial keratitis, scars which are the result of fissures about the mouth and marked thickening of both tibiæ. The liver is somewhat enlarged and the spleen palpable. Her whole appearance is that of a poorly developed, undernourished girl who at first appearance might be described as of habitus phthisicus. A large raised, reddish indurated papule measuring about 5 mm. in diameter is seen at the site of a luetin inoculation, performed forty-eight hours ago, and may be described as a papular form of positive reaction to the luetin test (Fig. 1, frontispiece).

In the following we are going to discuss the luetin reaction as introduced and described by Hideyo Noguchi, review the clinical results and statistics of the test, discuss the effect of administration of potassium iodide, make comparison between luetin and Wassermann reactions.

In 1911, Hideyo Noguchi,<sup>12</sup> of the Rockefeller Institute, described a cutaneous reaction for the diagnosis of syphilis, the reaction following upon injection of emulsion of dead spirochætae which he called Luetin.

He describes the cultivation of organisms for the production of luetin, as follows: Two strains of *Spirochæta pallida* were cultivated under anaerobic conditions for periods of six, twelve, twenty-four and fifty days at thirty-seven degrees C. One strain was cultivated in ascitic fluid and the other in ascitic fluid agar, each one containing a portion of sterile placenta. The lower portion of each solid culture in which a dense growth had occurred, was cut out and removed. The agar columns which contained numerous spirochætae were then carefully ground in a sterile mortar and the thick paste was gradually diluted by the fluid culture, which also contained many organisms. The dilution was continued until the emulsion became liquid. After heating the preparation for sixty minutes at 60 degrees C. on a water bath, 0.5 per cent. of carbolic acid was added. When examined under the dark field microscope, forty to one hundred dead spirochætae per field could be seen. This preparation

Noguchi named "Luetin." As control, a carbolized emulsion was made without spirochætæ.

The luetin was first applied by Noguchi to rabbits and when found positive in luetic rabbits, he applied it to man. He studied 400 human cases, 177 being syphilitic in nature, 77 of parasyphilitic nature and 146 as various controls. All the normal cases reacted negatively, that is, only a small erythematous area appeared at and around the point of injection and the reaction receded gradually between twenty-four and forty-eight hours, leaving no induration.

In cases of primary and secondary syphilis, with little or no treatment, no skin reaction occurred, except in a few instances. Most secondary cases which had been mercurialized before salvarsan administration and which remained without symptoms for some months after salvarsan injections, gave striking reactions. In manifest tertiary syphilis the luetin reaction was positive in all cases, in latent tertiary affections it was positive in 94 per cent. of all cases and in congenital syphilis in 96 per cent. of all cases.

Noguchi described three forms of positive luetin reactions:

(1) *Papular Form*.—A large, raised, reddish, indurated papule, five to ten mm. in diameter appears in twenty-four to forty-eight hours. The papule may be surrounded by a diffuse zone of redness. The induration increases slowly for three or four days and then the process recedes. The papule becomes dark blue and this disappears, as a rule, within one week. A trace of reaction, however, may persist longer in secondary lues under treatment or in congenital lues.

(2) *Pustular Form*.—Similar to the papular form until fourth or fifth day, then the inflammatory process progresses. The surface becomes indurated, the round papule becomes mildly edematous and multiple miliary vesicles occur through central softening of the papule, later the pustule ruptures, the margin remaining indurated. A crust forms and falls off and induration disappears. Hardly any scar is left. The pustules vary in size.

(3) *Torpid Form*.—The papular reaction diminishes to an almost invisible point in from three to four days, appearing negative, but lights up again after ten days and progresses to the pustular stage. This form of reaction occurred in a series of one of the authors (A. L.), once in the primary stage, once in the hereditary type and twice

FIG. 4.



**Congenital syphilis, showing Hutchinson teeth, interstitial keratitis, labyrinthine deafness and periostitis of tibiae.**



in the secondary stage, all of these cases having been under mercurial treatment.

With the exception of a slight rise in temperature in most positive cases, as a rule, there is no constitutional disturbance; only in three tertiary cases and one hereditary case there occurred general malaise, anorexia and diarrhoea.

Noguchi's research naturally led many men to try the luetin reaction on the patients. The results varied in the hands of different authors.

Fox<sup>13</sup> found luetin positive in 6 out of 14 cases of active secondary lues, or 42 per cent. of cases, while the Wassermann was positive in 100 per cent. Luetin was positive in 17 out of 33 cases of tertiary, or 51 per cent., the Wassermann was positive in 65 per cent. In 10 cases the reaction was equally marked on both arms, that is the reaction and control.

Rytina<sup>14</sup> considers luetin specific. He found it negative in primary and secondary untreated cases and positive in cases that received treatment. He found it positive in 100 per cent. of congenital latent and tertiary active, while in tertiary without symptoms it was positive in 94 per cent. and in parasyphilis in 80 per cent. of cases.

Alan Brown<sup>15</sup> thinks luetin specific in hereditary syphilis. In 100 normal cases tested 96 were negative and 4 showed a doubtful reaction. All of thirty-four syphilitic babies tested reacted positively to luetin.

Gordon,<sup>16</sup> in 22 cases of congenital syphilis, found 18 or 81 per cent. of positive reactions. All of these were over one year of age, and he believes that this is the minimum age limit for positive reactions.

Clauzaz<sup>17</sup> applied luetin in 71 cases of syphilis—10 suspects—and 30 non-syphilitic affections, comparing the findings with the Wassermann reactions. The positive response was decisive. The luetin was sometimes positive where the Wassermann was negative in cases of known lues. The torpid luetin reaction was at times delayed two weeks in developing.

Schippers,<sup>18</sup> however, made a rather unfavorable report on the luetin test. He has tried luetin in children, and his report speaks against the use of the test, at least in children. He has performed the

test on twenty children between the ages of three months to fifteen years, all of whom were afflicted with lues hereditaria. Of these, 11 cases gave papular reaction, 3 pustular reaction, 1 delayed reaction and 5 gave no reaction at all. Two of the children that gave negative results were in a very debilitated condition; one of them died two days after the test was performed, the other developed erysipelas facialis three days after. The other three children had been under treatment for a longer time, their condition of nutrition being good. Schippers claims that the technic of the test could not be responsible for the negative results.

Besides the cases of lues hereditaria Schippers has made control injection in 54 children between the ages of two months and eleven years, in 18 of these cases more or less distinct formation of the papule was present and twice even a pustule formation occurred. The papules, however, were not so darkly colored as the genuine ones, but in the majority of cases they must be regarded as doubtful reaction.

The diseases in which the reaction was positive, were: chronic enteritis (1, pustule), anæmia (1), tetany (1), psoriasis (1), chronic infantile eczema (4), exudative diathesis (3, 1 pustular), ulcer cruris (1), and healthy child (1).

Schippers comes to the following conclusions in regard to luetin test in children:

(1) The luetin reaction is not sufficiently reliable, about 16 per cent. of these cases were negative in lues, and about 5 per cent. in non-luetic cases.

(2) The test takes too long a time for interpretation—at least eight days.

(3) The reaction is negative in greatly debilitated luetic children (with infiltrated skin or oedema) and often positive or doubtful in children with exudative diathesis.

Of interest is the effect of potassium iodide, a drug which has been used so extensively in syphilis, on the luetin reaction. Sher-  
rick<sup>19</sup> tried the effect of potassium iodide on the luetin reaction in neurological cases and on the basis of his tests he comes to the following conclusions:

(1) A positive pustular or nodular luetin reaction can be obtained in 99 per cent. of all cases, irrespective of the presence of syphilis, by

the administration of potassium iodide, either simultaneously or shortly before or after the intra-dermal test.

(2) Other substances, such as agar and starch when injected intradermally will give a similar reaction when potassium iodide is administered. With these substances, however, potassium iodide must be administered within a shorter time than is the case with luetin.

(3) Other drugs containing iodine have a similar influence on the luetin reaction.

Kolmer and his co-workers<sup>20</sup> have come to similar results as to the influence of potassium iodide on the luetin reaction. They observed also a positive luetin reaction among normal non-syphilitic persons as late as one month after the ingestion of large doses of potassium iodide and in some instances the administration of potassium iodide caused the site of a former luetin injection to develop inflammatory phenomena and pustulation.

On the basis of his experiments Kolmer comes to the conclusion that "a positive luetin skin test has little value in the diagnosis of syphilis among persons who are taking or have recently taken potassium iodide. The amount of the iodide capable of producing these reactions varies considerably; also the length of time following the ingestion of iodide when this reaction to luetin may follow. For these reasons physicians should very carefully rule out the possible influence of iodides before conducting the luetin skin test."

De Buys<sup>21</sup> observed a very violent reaction in a mother and a nursing baby, in both of whom there was not the slightest evidence of syphilis. The mother, however, had some rheumatic pains for which she was taking fifteen grains of potassium iodide three times daily. The discontinuance of iodides caused a subsidence in the luetin reaction of both mother and child, and a return to the administration of the iodides to the mother produced again a positive luetin reaction in both. The iodides given in positive cases seemed to prolong the positive reactions.

*Luetin Compared With Wassermann Test.*—According to Noguchi luetin cutaneous reaction was more frequent in tertiary and latent forms of syphilis and Wassermann serum reaction more frequently positive in the primary and secondary stages of the disease. More-

over, the Wassermann reaction was more directly and immediately affected by the anti-syphilitic treatment than was the luetin reaction.

Kilgore<sup>22</sup> has performed the luetin test on 36 syphilitics and on 86 control cases. All of the control cases with the exception of two who suffered from diseases of joints gave negative reactions. Of 22 cases of tertiary and latent syphilis tested, 14, or 64 per cent., reacted positively to luetin and negatively to Wassermann. Of 12 cases of parasyphilis of cerebrospinal type 5, or 42 per cent., gave positive luetin tests and 9, or 75 per cent., positive Wassermann. There was 1 case that gave a positive luetin and a negative Wassermann. In 2 cases of congenital lues luetin was negative in both and Wassermann negative in one. Kilgore concludes that the Wassermann reaction is positive in a higher percentage of cases of late syphilis than is luetin and that the value of luetin is mainly in a small number of cases in which the Wassermann is negative and the luetin is positive (4 out of 36).

Pusey<sup>23</sup> is of the opinion that luetin is an important addition to the diagnostic methods of syphilis, since it is positive in some cases where the Wassermann is negative.

Gordon<sup>24</sup> thinks that the luetin test is more adaptable to paediatric work than Wassermann test.

An interesting report is that of E. B. Vedder and W. B. Borden,<sup>25</sup> who made comparisons between the Wassermann and luetin reactions in 744 cases in the United States soldiers who have served twenty years in the army, 80 per cent. of these cases being between the ages of fifty and eighty. Practically all of the inmates suffered from some disability, there were also many cases of locomotor ataxia. The Wassermann and luetin reaction did not give corresponding results. Out of the 744 cases 156 were positive to Wassermann, while 239 of this number gave a positive luetin. The luetin test, therefore, according to these authors, is more delicate as a routine test than the Wassermann. The luetin test was not obtained in cases where syphilis could be excluded. The Wassermann reaction, however, according to the authors, is much more constant in primary and secondary forms. In tertiary latent, and treated cases the luetin is considerably more efficient in detecting syphilis than the most efficient Wassermann reaction.

De Buys (*loc. cit.*) regards luetin as more valuable in hereditary syphilis than Wassermann test. Comparing these two tests he says: "We do not believe that luetin should displace Wassermann, as both tests, it would seem, serve distinct purposes, the Wassermann to give evidence of the presence of antibodies in circulation, indicating an active process, while luetin test not only gives this evidence, but also indicates an existing syphilitic condition, even though it be inactive."

*Conclusions.*—(1) The luetin test must not be regarded as being in itself sufficient for diagnosis of syphilis and is rather to be looked upon as confirmatory, since it occurs in a certain percentage of non-syphilitic cases.

(2) The chief value of the luetin test is diagnosis of syphilis in cases in which Wassermann is negative and luetin test positive, provided that the patient has not taken potassium iodide in large doses for at least one month.

(3) The luetin test is of considerable value in congenital syphilis and in tertiary and latent forms of the disease.

(4) The luetin test is devoid of any danger.

#### FOOD REACTIONS

Of eight cases of anaphylaxis to egg albumen which one of us (J. H. H.) has under observation, the one which we are showing here presents a most interesting history and illustrates the influence of heredity.

**CASE I.**—The grandmother of this eight months old infant presented no idiosyncrasy following ingestion of eggs, until the time of her first pregnancy, when during the fifth month she ate eight eggs in one day. Since that time, twenty-four years ago, she never has been able to relish eggs. Her first child was unable to eat eggs or anything containing eggs during her childhood, but during her later years she has been able to eat food containing a moderate quantity of egg.

Her second child, the mother of the present patient, gave the same history, stating that she could detect the smallest quantity of egg in pastry, almost at the first taste. Our patient has been a perfectly well infant until seen at eight months, when it was still exclusively breast fed, and had developed a severe dyspepsia. For

twelve hours it was placed on barley water, with instructions to add the white of one egg to a pint of barley water during the subsequent twelve hours. It partook of one ounce of this mixture of barley water and egg albumen. Within thirty minutes it became violently ill, with vomiting and purging, and shortly thereafter developed marked œdema of the entire body. This lasted for about six hours, when it receded spontaneously. When a slight abrasion was made with a Von Pirquet scarifier and a drop of egg albumen applied to the abrasion, a white wheal one-half inch in diameter developed within six minutes, increasing in size up to fifteen minutes when it was surrounded by an erythematous area, one inch in diameter, and sprinkled throughout this erythematous area, numerous pin-head sized white elevations developed. The entire reaction disappeared in one and a quarter hours.

This description is typical of the reaction obtained in this class of infants, some reacting less markedly and less rapidly than others.

It has long been known by many clinicians that there are a certain number of children who are made ill by ingestion of a small quantity of egg. The mothers very often pay little attention to it except for the avoidance of egg in their children's diet.

Oscar Schloss<sup>26</sup> has done extensive research on this subject. He injected egg albumen into the skin of children that were supposed to be susceptible to egg and found that an urticarial wheal developed at the point of injection in five to fifteen minutes after the injection. The size varied from 1.5 to 3 cm. in diameter. He used egg white in different dilutions. In 1:100 dilution a marked reaction occurred. He later separated from the protein the protein free elements, and no reaction occurred, showing that the protein part of the egg was the exciting cause. Of this the ovomucoid was the most active, producing results in 1: 15,000 dilution, and sometimes even in 1: 20,000 dilution. Ovomucin and ovoglobulin were next in activity. Ovoalbumen was active only in a comparatively concentrated solution. Egg yolk in 1: 15 dilution produced a reaction. This experimenter has also produced immunity to egg in cases susceptible to it. He started with small quantities of ovomucoid and gradually increased the dose. The reaction became gradually weaker and then disappeared. The child was then able to take eggs without any discomfort following. This

observer considers the idiosyncrasies to egg not a congenital affair, but one acquired between ten days and fourteen months of life. H. M. Rich,<sup>27</sup> on the other hand, considers heredity a strong factor. He found in 5 out of 15 cases reported a family tendency to egg idiosyncrasy. Of late several authors emphasized the role of food-idiosyncrasies in the production of asthma in children.

*Technic.*—The skin tests to food substances may be performed either by cutaneous inoculation or intracutaneous injection. The intradermical method is more sensitive and at times may prove positive, when the cutaneous test is negative, resembling thus the tuberculin cutaneous and intracutaneous tests in their sensitiveness. In our own work we have found that the application of a drop of fresh egg albumen followed by the abrasion of the skin by the Von Pirquet borer was sufficient to produce a positive reaction in most instances within five to six minutes.

While the test can be made with foods, such as milk and egg in unaltered state, it is of advantage to use the proteins in comparatively pure state, when they can be obtained in such form. The latter, however, offers an obstacle to the more general use of the tests.

*Reaction.*—The positive reaction, as generally seen following cutaneous test, results in early development of an urticarial lesion which passes through more or less definite cycle. A white wheal varying from one-fourth to one-half inch or more in diameter usually develops within five to six minutes and increases in size up to fifteen or twenty minutes. By this time it is usually surrounded by an erythematous area, an inch or more in diameter. In the case of the reaction to egg albumin, numerous small pin-head sized white elevations are seen sprinkled throughout this erythematous area. The entire reaction usually disappears within one or two hours.

Frequently following the intradermal method a reaction inflammatory in character is seen after five hours which increases in size for twelve to twenty-four hours.

*Interpretation.*—A positive reaction almost without exception indicates an idiosyncrasy to this particular food substance. A negative skin reaction, however, does not necessarily exclude an idiosyncrasy, as it was found that in some cases in which the ingestion of certain food caused nausea, vomiting, diarrhoea and disturbance of

gastro-intestinal tract, the cutaneous test was negative. The reaction may also become temporarily negative following the stage of intoxication after ingestion of a food. In these cases, however, the reaction again becomes positive.

*Pseudo Reactions.*—Pseudo reactions are sometimes seen in infants with sensitive skin, more especially those suffering from an exudative diathesis. Such cases can usually be differentiated by obtaining the same reaction with other foods which do not result in acute intoxication when ingested.

*Other Food Reactions.*—Similar reactions have more recently been described in individuals with an idiosyncrasy for cow's milk, cereals, vegetables, fruits and meats.

*Conclusions.*—(1) The cutaneous test is easily applied and in most instances makes a positive diagnosis of the disturbing food.

(2) To make a diagnosis as to the specific protein involved, the individual proteins must be obtained in pure state.

(3) Some of the cases are sensitive to more than one form of protein.

(4) In some of our cases the idiosyncrasy appeared to be hereditary, in others it appeared to follow sensitization to repeated ingestion of certain foods.

#### REFERENCES

1. PARK AND ZINGHER: Active immunization with diphtheria toxin-antitoxin. *J.A.M.A.* lxv, 2216.
2. SCHICK, B.: Kutanreaktion bei Impfung mit Diphtheriatoxin. *Münch. Med. Wochschrift.* 1908, iv, 504.
3. COWIE, D. M.: Observations on the intradermal and repeated intradermal injection of diphtheria toxin with reference to the Schick test. *Am. Jour. Dis. Child.* 1916, xii, 266.
4. ZINGHER: Methods of using diphtheria toxin in the Schick test and of controlling the reaction. *A. J. Dis. Child.* xi, 1916, 269.
5. PARK, ZINGHER AND SEROTA: Schick vaccination and its practical application. *Arch. Ped.* 1914, xxxi, 481.
6. VEEDER: *Am. Jour. Dis. Child.* 1914, viii, 154.
7. MOFFET, R. D. AND CONRAD, A. C.: Observation on the intracutaneous reaction of Schick. *J. A. M. A.* Sept. 18, 1915.
8. MOODY: *J. A. M. A.* 1915, lxiv, 1206.
9. KOLMER, J. A.: An anaphylactic skin reaction to diphtheria bacilli. *Am. Jour. Dis. Child.* 1916, xii, 316.
10. SCHICK (*loc. cit.*).
11. LEVINSON AND BLATT: Studies in the Schick diphtheria reaction. *Archives of Diagnosis.* 1915, viii, No. 3.

12. NOGUCHI, H.: A cutaneous reaction in syphilis. *Jour. Exp. Med.* 1911, xiv, No. 6.
13. FOX, H.: Experience with Noguchi's luetin reaction. *Jour. Cut. Dis. incl. Syph.* 1912, xxx, 455.
14. RYTINA, A. G.: The luetin skin test in the diagnosis of syphilis. *Med. Record.* 1913, lxxxiii, 384.
15. BROWN, A.: The luetin reaction in infancy. *Am. Jour. Dis. Child.* 1913, vi, 171.
16. GORDON, M. B.: The value of the luetin reaction in congenital syphilis.
17. CLAUSZ: Diagnostische Versuche mit Luetin-Noguchi. *Münch. med. Wochschrift.* 1914, lxx, 1933.
18. SCHIPPERS, J. C.: The value of luetin reaction in pediatrics. *Zschrft. f. Khlk.* 1915, xii.
19. SHERRICK, J. W.: The effect of potassium iodide on the luetin reaction. *J. A. M. A.*, lxv, 404.
20. KOLMER, J. A., MATSUNAMI, T., AND BROADWELL, S.: The effect of potassium iodide on the luetin reaction. *J. A. M. A.* 1916, lxvii, 718.
21. DE BUYS, L. R. AND LANFORD, J. A.: A comparative study of the luetin and Wassermann reactions in infancy and childhood. *Am. Jour. Dis. Child.* 1916, xii, 387.
22. KILGORE, A. R.: Luetin cutaneous reaction for syphilis. *J. A. M. A.* 1914, lxii, 1236.
23. PUSEY, W. A., AND STILLIANSON, A. W.: Noguchi luetin for syphilis. *Jour. Cutan. Dis. incl. Syphl.* N. Y., 1914, xxxii, 560.
24. GORDON (*loc. cit.* 5).
25. VEDDER, E. B., AND BORDEN, W. B.: A comparison of the Wassermann and luetin reaction in 744 individuals. *J. A. M. A.* 1914, lxiii, 1730.
26. SCHLOSS, OSCAR: Allergy to common foods. *Am. Jour. Dis. Child.* iii, 341.
27. RICH, H. M.: Infantile sensitization to egg-albumen. *Jour. Mich. Med. Soc. Grand Rapids*, 1914, xii, 649.

CLINIC  
AT THE LYING-IN HOSPITAL

By ROSS MCPHERSON, M.D.

New York, April 30, 1918

---

I. Endometritis following abortion	III. Perineorrhaphy .
II. Incomplete abortion	IV. Median forceps operation

OUR afternoon work consists of four cases. The first is a curettage for endometritis following an abortion occurring some years ago. The second is an incomplete abortion. The third case is a perineorrhaphy, and the fourth a median forceps operation.

The first two cases are a happy combination, as they serve to illustrate most beautifully the proper way of caring for a miscarriage. If we had hunted for a long time for examples to illustrate this point it is doubtful whether we could have found two cases as well suited to this purpose.

The first case will illustrate the improper way of caring for a miscarriage and the second will show what we consider the best method of caring for such a case. We divide abortion into three groups: *threatened*, *inevitable*, and *incomplete*; and since we have treated such a large number of cases in this institution we feel that our opinion ought to be worth something as regards the method and technic of handling them. There is at the present time a great deal of discussion as to the proper method of handling cases of miscarriage. There is a general agreement that in cases of *threatened abortion* it is better to put the patient to bed, keep her quiet, administer opiates, and make an effort to carry her through to term. Then after a few days she is allowed to be up and if no more bleeding occurs, the case is considered arrested. On the other hand, there is considerable difference of opinion as to which is the better course to pursue when one gets a patient with more or less hemorrhage, cramps, more or less dilatation, and perhaps the uterine contents protruding from the os. This is where there is a divergence of opinion among obstetricians. One group favors leaving the patient alone, keeping her quiet, and perhaps the administration of pituitrin, believing that she will expel the uterine contents herself without any interference on the part of the obstetrician. The other group, of which I am an

enthusiastic member, believes that when one has made up his mind that a patient is going to miscarry, or in other words, that miscarriage is inevitable, it is best to empty the uterus in a surgical manner, that is, of course, if there is no infection. If the os is so fully dilated that we can empty the uterus at one sitting well and good, but if we have to wait for the uterus to empty itself, the proper procedure is to pack it with iodoform gauze with the patient under anesthesia. Then we give the woman twenty-four hours in which to dilate. At the end of that time, under anesthesia and careful asepsis, the packing is removed and the uterus curetted. Whether this is done by means of the gloved finger, the dull curette or the sharp curette, depends upon the man who is doing the work, upon what instrument he likes and can use most skilfully. Personally I prefer an instrument to the finger. We can sterilize instruments thoroughly and they are likely to be cleaner than the finger. The use of the bare finger I would consider criminal malpractice.

My procedure is to remove the packing and then with exactly the same aseptic care as in a laparotomy, go over the operative field with iodine and then to use a sharp curette to remove the remainder of the decidua. This is the method employed by practically every one treating these cases of miscarriage in this hospital, except that some of the men use a dull curette. Some object to the curettage on the ground that there is danger of infection, especially in cases that they think have expelled the entire uterine contents. We, however, have the theory that there is no such thing as a complete miscarriage and we go through this same procedure in all cases, immediately if the uterus is sufficiently open; if not we pack and wait until the next day to do the curettage. The result of this procedure is that we practically never lose a case. I do not know when we have lost a case, and we have had five, six, seven, or possibly eight thousand cases since I have been connected with this institution. In 1905 I reported 2,500 cases and we have been having many since that time, and our results have been uniformly and absolutely good.

#### ENDOMETRITIS FOLLOWING ABORTION

**CASE I.**—This patient had a miscarriage about one and one-half years ago, and so far as I know it was not a criminal case. She has never been well since. Under the advice of her physician at the

time of her miscarriage she stayed in bed for a few days and then went along without further attention. Ever since she has had a leucorrhœa so severe that she has had to wear a pad and has been very uncomfortable. Examination shows no tenderness in the region of the uterus or ovaries. Smears from the vagina and cervix are negative for gonococci as is also the complement fixation test. There is nothing to prove that the patient has ever had a gonorrhœal infection. From the history it may be assumed that the case is one of neglected miscarriage. Some of the placenta probably remained in the uterus, which led to the development of an endometrial hyperplasia, which does not seem to have involved the cervical glands. I think under these conditions we are justified in doing a curettage, and in hoping that the discharge will thereby be stopped. There is nothing that is more difficult to cure than these cases of leucorrhœa.

We use Hegar dilators instead of the Goodell dilators. There is no particular reason for this except that with the former we have obtained excellent results. In using Hegar's dilators we must be careful not to perforate the uterus, and on this account we make downward traction on the cervix, thus bringing the uterine canal into a straight line. As soon as this woman has been curetted she will get along very comfortably; she has had no menstrual difficulties at all.

In doing a curettage I believe that it is necessary to employ a sharp curette. There are many who object to the use of the sharp curette because they say it breaks down the barriers to the entrance of infection. That may be true of infected cases, which should not be curetted at all. It has been my experience that if one wants to do a thorough piece of work he needs a sharp instrument. I am willing to grant that a sharp curette is a dangerous instrument, but so is a sharp knife or a sharp pair of scissors; these are all dangerous instruments in the hands of a tyro. I find that I can curette with a sharp instrument in a soft uterus without doing any harm, if I use the instrument gently.

I like to wipe out the uterus with iodine. Possibly this is a procedure which only salves my conscience. You may recollect that there was a carbolic age, a time when we thought we must use carbolic acid. Then there was a bichloride age, when we thought we must use bichloride, and now we are having an iodine age. All the

claims now made for iodine were made in times past for carbolic and for bichloride and it is possible that iodine does no more to prevent infection than did the other substances.

I usually put in a little iodoform gauze to irritate the cervix and to excite contraction. That is removed in two or three days and we hope that the patient will have no further trouble.

#### INCOMPLETE ABORTION

CASE II.—There may be many things in which the opinion of any obstetrician is just as good as mine, but I do not believe anyone gets any better results in the treatment of this type of cases than we do in this institution, and hence I think our opinion is entitled to a great deal of consideration. I know of one man for whose opinion as an obstetrician I have great respect, who says he never does anything for these cases, but just lets them alone, and that he never meets with a severe hemorrhage. He must get a different class of patients from those we get here because we do have cases with severe hemorrhage. Some practitioners may get only ten, fifteen or twenty such cases in a year, while here we are getting them all the time and that may account for the difference in our opinions. But we do get cases with hemorrhage, and packing with gauze lessens the hemorrhage and accordingly we employ it. Some give the various glandular extracts and believe they are effective. I do not know that they do any good, but at least they do no harm. This is not only an iodine age, it is the age of glandular extracts. I recently had one sample left at my office that contained pituitrin, adrenalin, ovarian extract, pancreatic extract, lutein, thyroid, and the extract of practically every ductless gland, all mixed together; such a mixture is about analogous to the old shot-gun prescriptions which we used to see and hear of.

To come back to our case—this patient, who was between two and three months pregnant, started to bleed and had quite severe hemorrhage. She was packed by her physician at home and then came in here. We found the packing had not been properly done, was protruding from the vulva, and that there was not any in the cervix at all. We packed her properly and before doing it took cultures from the cervix and vagina. She has now been having some pain for quite a little while and I hope that on removing the

packing I shall find the cervix dilated so that we can get into the uterus without any trouble. I shall remove the contents of the uterus and scrape it very lightly with the curette and then pack it. If the uterus is cleaned out thoroughly it need not be packed, but it does no harm to put in the packing. I feel perfectly convinced that when this method is carried out with proper attention to the details of the technic it offers distinct advantages over the old way of allowing these cases to take care of themselves. I heard a paper read recently that took the ground that curettage is practically criminal, and I agree that as practiced by some without administering an anæsthetic and without surgical asepsis curettage is wrong. It is better to leave the patient alone than to attempt to perform a curettage in the patient's home without an anæsthetic and without proper surgical asepsis.

While we are waiting for the next patient I would like to call your attention to the gray operating room and the gray dressings. Doctor Markoe some years ago visited the Peter Bent Brigham Hospital in Boston and found that they had their operating room finished in gray and were using gray dressings, and he was impressed with the idea that this color offered certain advantages over white and was a very good thing, so the plan was adopted here. We find that it is very much easier on the eyes of the operator and those of his assistants than the white. It is also better for those who witness the operation, as one can see what is being done much easier, as there is nothing to distract one from what is being done, but when one is surrounded by white there is an inclination for the eye to follow it. Another advantage is that the gray looks cleaner, as it does not show wear and tear as readily as does the white, and it can be kept clean with less labor than the white, which is an advantage at this time when it is so difficult to get employes. At the Massachusetts Institute of Technology a study was made of the effect of different colors in the operating room and it was found that a tobacco brown was the best color, then a gray, next dead white and that the very worst color one could have was a shining white, yet it was the shining white that one most always saw used in operating rooms. Unfortunately, it is now almost impossible to get the gray cloth, as it is very expensive and does not wear well. So while the gray may

be best on theoretical grounds, we may be forced to abandon its use in the near future.

We now have our patient, who as I told you, was packed last night. We will first remove the packing and see what has happened. There is a little bleeding and she has had considerable pain. The os is well dilated and the foetus can be seen. The sac is protruding down into the vagina. I will grasp the anterior lip of the cervix with the sponge forceps to avoid tearing the tissue as is the case with the volsellum forceps. I will then run the sponge forceps around in the uterus and get out anything that is there. Next we take the sharp curette and run that around lightly. You may see that I am not working very hard with the curette; I am just using it gently. The uterus is sufficiently open so that I can take the gloved finger and pass it around the uterus and see that I have gotten it clean, which it now seems to be. There is another point and that is that as the uterus begins to contract it will do no harm to put in packing and just a little iodine so that we can feel that we have taken every precaution against infection. Now, if we have left anything in the uterus it will come out of itself on the gauze when it is removed. We put the packing in right up to the fundus, and we will leave it in until tomorrow. Now we know that the uterus is clean and just what condition we were dealing with and I submit to you that it is better business than leaving something behind or not knowing with what condition one is dealing. This woman has not been under the anæsthetic more than ten or twelve minutes and she will make an absolutely uneventful recovery, provided she has not been tampered with and infected before she came here.

#### PERINEORRHAPHY

**CASE III.**—Our next case is a plastic operation for a laceration of the perineum some four months post partum. We will use the so-called Mayo operation, which is a modified Lawson Tait. In doing perineal repairs we have given up the old-fashioned denuding operations, which required an elaborate armamentarium, a variety of curved scissors, etc., and by which we did a lot of clipping of little bits of tissue. I have not done that kind of an operation for a long time. We used to do a perineorrhaphy of that kind, and when we were through we found that it had taken an hour and one-half and

we had succeeded in doing a gynaecological stunt, but we had not achieved correct anatomical results and the operation did not fulfil the purpose for which it was designed; we did not get the muscles together. In order to obtain the correct anatomical results we must approximate exactly muscle to muscle, fascia to fascia, and skin to skin. The men who operated by the old method fifteen or twenty years ago lost sight of this fact in their endeavor to secure a beautiful cosmetic effect. They obtained a result that appeared good at first, but as they did not get the muscles together after five or six months the perineum again flattened out and the patient was no better than before her operation. The Lawson Tait operation aims to secure a perfect result by matching the skin but not the muscles, and therefore fails. The first time I saw this operation performed which I am about to show you, was by Doctor Judd of the Mayo clinic. It appealed to me as simple and as fulfilling the anatomical requirements and so I have been using it ever since. Now almost everyone is doing this operation and the results are perfect and anatomically correct. Another point in favor of this operation is that it is rapidly performed. It only takes about ten minutes to do a perineorrhaphy in this way whereas it used to take an hour and a half by the old method. The results after this operation have as a further point in their favor the fact that the result is permanent. I have cases in which it was performed seven or eight years ago and they are in perfect condition at the present time. Again, anyone with even a slight degree of surgical ability can do it and he does not have to have an elaborate armamentarium; it can be done with any ordinary set of instruments.

In performing the operation, as I am now showing you, the whole trick is to get the line of cleavage. The first step is to clip off the edge of the laceration with a knife or scissors and then get the line of cleavage. Having found the line of cleavage on both sides we then, by blunt dissection, free the muscles for the extent of the laceration, being careful not to perforate the rectum. For this purpose I use an open gauze sponge and find it very useful. After going pretty well back on either side we then take the forceps and pick up the muscles on either side and approximate them the one to the other. For this purpose I use No. 3 plain catgut, putting in two catgut sutures. Then in order to get a good cosmetic result

we first do a crown stitch which starts at the angle and goes around the wound, puckering up the edge of the post marginal wall and when we get back to the angle where we started we tie the whole thing. We must be careful of one point and that is to eliminate the dead space away back in the wound. I like to tie the upper crown stitches first so that we have something to work from. We now know that as far as any results are concerned the operation is complete excepting that the skin has not been closed. In closing the skin we use a subcuticular stitch, so that when the wound is healed there is no scar, the patient having a perfect perineum and a perfectly correct anatomical result; one who was not told of the operation would never be able to tell that the patient had been operated upon. In doing this we have been exactly eight minutes. We have left muscle to muscle, fascia to fascia and skin to skin, and we have secured the result for which the operation was undertaken, which is the only justification for performing it.

#### MEDIAN FORCEPS OPERATION

CASE IV.—This patient has been in labor for a long time. Her systolic blood-pressure is 170 and she has considerable oedema. I think it is better both for the baby and the mother to deliver her at once.

In doing a median forceps operation there are several primary points to remember in the application of the forceps. Many text-book rules are not good, but those given for the employment of forceps are correct and should be followed. In the first place, never put the forceps on when the os is undilated or undilatable. Never put the forceps on where the membranes are unruptured. If the membranes are unruptured, first rupture them. Never put the forceps on until you are sure of the position of the child. Rarely, if ever, apply the forceps on a dead head. If you follow these rules carefully and conscientiously you will have very little trouble provided you understand the cardinal points in the use of the forceps. It is not an instrument to be used like a corkscrew, which seems to be the idea that many have. It is a dangerous and ill-devised instrument, but still it is the best we have for the purpose of extracting a living baby from a living mother. Our object in using the forceps is to accomplish this end in a manner as nearly approaching that of a normal birth as possible.

Having decided that the baby must be delivered and that the forceps must be used in extracting the baby, the next thing to do is to determine the position of the head and the amount of dilatation of the cervix and whether the membranes have been ruptured. If the membranes have not been ruptured, the obstetrician should rupture them and then it may be well to give the patient a little more time, for possibly after the membranes are ruptured she will deliver herself. Before applying the forceps bear in mind the danger of injuring the child's head and of increasing the laceration of the mother. With these points in mind one may proceed to apply the forceps and extract the baby; and in the ordinary case there is not much trouble in effecting a delivery.

The forceps is an instrument made up of two curves, a pelvic curve and a cephalic curve. The common way of putting the forceps on is to insert the right hand into the vagina and then to apply the left blade of the forceps to the head with the handle pointing toward the right thigh. There is no question that one can get the instrument on that way, but there is a better way devised by Doctor Markoe, who is a man of an extremely mechanical turn of mind, and in making manipulations is always thinking of the mechanics of the problem. It occurred to him that instead of trying to adjust the pelvic and the cephalic curves of the blade at the same time that it would be better to give attention to one curve at a time, especially as it was difficult for most people to think of and do two things well at once, so he devised this method of putting on forceps which I am going to demonstrate. By applying the blades in the way I am going to show you the procedure is very simple. With the right hand in the vagina the forceps is introduced in the median line so that one can apply the right thumb to the edge of the blade, holding the handle lightly with the left hand and dropping the blade gently into the vagina with the shank against the symphysis, now bringing the whole instrument downward in the midline. When the tip of the forceps touches the head one may begin to take an interest in the cephalic curve and rotate the blade so that it will engage on the head. This movement is mechanically correct both as regards the adjustment of the pelvic curve and the cephalic curve, both being done as two separate motions. The other blade is then applied in the same way.

I have a rule that if anyone sees me make an error as regards technic in the operating room he or she shall call my attention to it at once, for if one is aware of such a break at the time it is made, it may be repaired immediately. Not to do this is, according to our code, not good manners in the operating room.

It is a good plan always to catheterize the patient just before applying the forceps, because we want to get all the room we can and if the bladder is full and presses downward there is great danger of injuring it and producing a vesico-vaginal fistula. The first thing we do after the patient has been catheterized is to examine for the position of the foetus. In this case the perineum is somewhat oedematous and in order to facilitate the examination I will lubricate my hand with a little sterile soap. The position is vertex L. O. A. This woman has two very sharply defined ischial spines. The pelvis is more of the male type, and that is the kind of a pelvis that puts more strain on the perineum so that it may be necessary in this case to do an episiotomy.

Now with the right hand in the vagina, we will introduce with the left hand the forceps blade, carrying it in with the handle in the median line and starting with it against the symphysis. The thumb of the right hand is against the blade to guide it, until the tip impinges on the head, the handle always remaining in the median line. As soon as the tip of the blade touches the vertex the handle is rotated toward the mother's left thigh and immediately the instrument passes around the head. Simultaneously the handle, still in the median line, is brought downward toward the floor and the instrument is in position. The other half is then applied in a similar manner, simply reversing the hands. I try to have every house officer learn this method of applying the forceps before he goes out into practice. Occasionally the instrument may need a little adjustment, but this is not usually necessary. In making traction it must be made downward toward the floor until the occiput comes under the symphysis. If we do not make the traction in this direction it is a question which is the strongest, the operator, the symphysis or the head, and the symphysis will stand considerable force. As soon as the occiput comes under the symphysis raise the handles slowly, and make outward traction.

A forceps case requires the very slightest amount of anaesthesia

and if the patient is not altogether under the anæsthetic and has several good pains it may help matters along and expedite the delivery. A version, on the other hand, requires deep anæsthesia.

We exert traction on the forceps and then we relax it and listen to the fetal heart. Then we again exert traction.

Now in this case the question of episiotomy comes up. It is almost impossible to do a great deal of forceps work without getting a higher percentage of lacerations than in normal deliveries, and this is especially true in primiparæ, among whom there will be a definite amount of laceration in almost every case. The question comes up whether it is better to have the laceration and allow it to run off irregularly in any direction or to cut down in the middle of the perineum and get a clean, straight wound that one can sew up easily. For years I have been loath to do an episiotomy. I would have a case in which I might do an episiotomy, and in which it would be a toss-up whether or not there would be a laceration, and I would decide to take the chance, with the result that I would get another laceration and would have to sew it up. About a year ago I decided to cut the perineum in a case that had been tedious and as soon as the perineum was cut the head almost fell out. I sewed up the wound and got a very good result. I do not think that one should do an episiotomy on every forceps case, but I think that we should do many more than we have been doing in the past. By doing an episiotomy one can sew up the tear easier and there is not so much destruction of tissue. If a patient has been in labor for hours and one finds that the head comes down pretty far and then disappears from sight again either there is a constriction ring and when one makes traction the whole uterus swings downward and then swings back when the traction is released, or else there is a short cord, or else the cord is around the neck. Under such conditions one should find out whether the cord is around the neck and if it is it should be released or if that is impossible it should be cut. I recently had a case in which the cord was very short, being only eight or ten inches in length. I put on the forceps and brought the baby out and pulled the placenta off with it. If I had not had everything in readiness to stop the hemorrhage I would have lost that case.

Another point I wish to emphasize is that in most cases a forceps

operation takes time and requires patience and one should not try to effect the delivery too rapidly.

On making the episiotomy incision one usually makes it about three-quarters of an inch long. Have the head well down so that the perineum is put on the stretch and then make a straight cut in the median line. You will find that then the head will advance very rapidly because you have removed the perineal resistance. Then as the head advances, if the cord is around the neck we just push it off over the head or cut it and deliver the anterior shoulder and then the posterior shoulder and the after-coming body. As the baby is delivered tip it upside down, keeping the head down so that it will not attempt to make an inspiration before you are prepared to have it do so. The assistant follows the fundus well down without exerting pressure.

Do not tie the cord until the pulsation has stopped, because by waiting until the pulsation has stopped the child gets additional blood which is a great advantage to it. After the baby takes a good inspiration and the pulsation has stopped then cord should be tied and a dry dressing applied to the stump.

Now we come to the least interesting and least understood part of labor, the third stage. We are likely to think that now that the child is alive and the mother delivered and is all right, everything is happily over and there is nothing to do but to deliver the placenta.

The third stage, however, is very important because it is at this time that we get the retained placenta, the postpartum hemorrhage, and it is the time when we discover the lacerations or else failing to discover them leave them to cause trouble later on, and then the other man discovers them. This is the stage during which one notes abnormalities in the cervix, sometimes fibroid of the uterus; it is the stage which if handled properly will result in a happy outcome of the labor in all respects, but if handled improperly all will go wrong. If I ask a class of undergraduates when one should remove the placenta, someone will answer, "In twenty minutes after the delivery." When the class sees that I am not satisfied with that answer, someone else will say, "In one-half hour." When they see that I am still not pleased with the answer, someone perhaps, will say, "When the uterus is fully retracted and the placenta is separated." That is the correct answer.

In this case there is considerable bleeding and the first thing to do in this and every case is to prevent bleeding by stimulation until the uterus has contracted. If we try to remove the placenta before that time we will have a hemorrhage and trouble. Watch the fundus until the placenta is separated, but do not use the Crede method until the placenta has separated from the fundus.

In this case we have an episiotomy wound and I want you to see how beautiful it is and how easy it is to close it. All we have to do is to join muscle to muscle, fascia to fascia, and skin to skin. By doing this we have saved lacerations and have obtained a very satisfactory result.

## CLINIC FOR MEDICAL OFFICERS OF THE ARMY AND NAVY

By JOHN F. ERDMANN, M.D.

AT THE POST-GRADUATE MEDICAL COLLEGE AND HOSPITAL, NEW YORK,  
MAY 3 AND 6, 1918

- I. Appendicitis
- II. Exploratory laparotomy (malignant disease of the sigmoid)
- III. Malignant disease in the upper abdomen
- IV. Enlarged cervical lymph-nodes
- V. Acute appendicitis
- VI. Herniotomy; excision of varicose veins
- VII. Excision of cervical lymph-nodes
- VIII. Fibroid uterus
- IX. Thyroidectomy
- X. Imperforate hymen
- XI. Intrahepatic stones
- XII. Exploratory laparotomy; duodenal ulcer vs. appendicitis
- XIII. Carcinoma of cæcum

**CASE I.—*Chronic Appendicitis.***—This patient is a woman, thirty-three years of age, who for the past two or three months has complained of attacks of indigestion and gas eructation, which have no relation whatever to the ingestion of food. She has pain in the right lower quadrant and tenderness over McBurney's point. A differential point between duodenal ulcer and appendicitis is that in appendicitis the pain and distress have no relation to the ingestion of food, while in duodenal ulcer the distress comes on from one to four hours after taking food, and is evidenced by pain, eructations of gas and discomfort, which is relieved by the administration of alkalies or by taking food. In appendicitis food seems in no way related to the symptomatology. In a young woman between sixteen and twenty-five years of age, who gives evidence of pain in the right lower quadrant, one cannot be too certain of the diagnosis of appendicitis, for in many such cases diagnosed as appendicitis one finds at operation a corpus luteum cyst.

In making the initial incision for appendicitis I have for many years followed the method of Kammerer. This incision has the merit of allowing one to extend it upward or downward, thus giving free access to the upper as well as the lower quadrant of the abdomen. Or we may make Deaver's incision, which is just over the rectus,

through the anterior sheath and rectus. We carry the incision down to the peritoneum, incising the transversalis fascia and the peritoneum at about the mid-rectus area. The first thing we do is to palpate the adnexa. The uterus is normal, but there is a very large right ovary, and a number of cysts follicular in character, one of which we will excise. As I told you, I made the Kammerer incision for no other reason than for the purpose of examining the adnexa. Having removed the ovarian cyst, I will now sew the denuded edges together with No. 1 catgut, to act as a haemostat. The hemorrhage is checked by the placing of these sutures. Some gynaecologists who see an ovary like this attempt to remove it and frequently do remove it. Personally I have tried to preserve ovaries of this sort, merely excising the cyst. We will now palpate the ovary on the other side. This ovary is apparently normal.

I would like to call attention to the appendix, which is large and moderately distended, the kind of an appendix formerly known as an open appendix and a mobile cæcum, "*Cæcum mobile*." We will transfix the meso-appendix, cut it at the point of transfixion, crush the base of the appendix and remove it. We will then ligate the meso-appendix, and after we have done this we will ligate the base of the appendix. You may recall that in the earlier years Dawbarn first advised that the open-mouthed stump of the appendix be inverted. Personally, I have had hemorrhage with two open-mouthed appendices. One of these so alarmed me that I have never since left an open-mouthed appendix stump, but always invert the stump after ligating. Perhaps those of you who are hypercritical will ask why, on theoretical grounds, we should invert the stump of the appendix, which might be infected and result in a concealed abscess, and even in a perforation of the intestine. I have done thousands of cases, inverting the stump in this way, and have had no trouble whatever.

In this case I am not going to do anything with this long movable cæcum; it will take care of itself. We will close the layers of the abdominal wall just as they were cut.

This patient will be up in from four to six days and will be out of the hospital in ten days. When she is put to bed she will be given one-sixth of a grain of morphine just as soon as she comes out of the anaesthesia, and one-eighth of a grain every three or four hours

for the first twenty-four hours. Now we will go back and sew up the fascia, or aponeurosis, or anterior sheath of the rectus, whichever you prefer to call it.

The appendix which we have just removed is pathological in the sense that it is enlarged, distended and has a large open mouth at its base, which would permit intestinal contents to enter it and this would be followed by spasms produced by attempts at expulsion of contents and later there might be concretions, ulcerations, and a typical gangrenous appendix, or an acute infective attack.

*CASE II.—Exploratory Laparotomy (malignant disease of the sigmoid).*—This woman is sixty years of age. She has not as complete a history as should be recorded. She has been losing weight and passing blood from her bowels. She complains that she feels as though there were some foreign body in her left side.

Rectal examination is absolutely negative. The abdominal examination shows a mass in the left iliac zone more than likely connected with the sigmoid. The patient's family has been told that the exploratory operation might lead to one of two results. There might be merely the exploratory operation and nothing else, as an inoperable malignant condition might be present, or it might be that an artificial anus would be required, or again it might be that the growth could be removed and an anastomosis of the intestines be made.

The patient has had some symptoms, such as pain, cramp-like in character and a discharge of slime and mucus, which rather points to a malignant condition of the bowel. Since she has had the anæsthesia I find that there is a hard mass in the naval which is evidently a metastasis from the growth in the abdomen. This metastasis gives us little to hope for from an operation. On the left side there is a movable mass; on the right side there is nothing definite, but I think there are a few enlarged glands. The liver shows no evidence of enlargement or nodulation. The first thing to do is to open the abdomen on the left side at a point corresponding to that point on the right side at which we make the incision for the removal of the appendix. The incision we make will be considerably longer, however, and will split the rectus muscle so that if we decide to establish an artificial anus the rectus muscle will act somewhat as a sphincter.

After the abdomen is open the first thing to do is to examine

the liver and the glands about the bowel with reference to metastases. There is a great deal of fluid in the peritoneal cavity so that the omentum seems to float. The fluid is a peculiar clear fluid, which is a sign of malignancy in this case. The nodule in the naval is not connected with anything at all. There is a large cauliflower growth on the fundus of the liver and one on the posterior aspect the size of a silver quarter. The omentum is very badly involved. In the left lobe there is a mass the size of a tangerine and in the sulcus there is another mass the size of an English walnut. It is difficult to say whether the mass in the sigmoid is the primary mass or not, but I think the primary growth is in the sigmoid, involving the intestines and omentum, and going down into the pelvis where it is attached to the fundus of the uterus. There is no use in taking a specimen, for the condition is very evident and there is only one thing to do and that is to close the abdomen and give the woman morphine when necessary for her comfort. I have no objection to the X-ray being tried, but I am satisfied that it will not be of any use except possibly to relieve pain. It is a waste of time and energy to use the X-ray or radium in a case of this sort unless it is done from the experimental standpoint.

Why was this growth in the sigmoid not discovered earlier? Because the only way it would have been discovered would have been to look for blood or mucus in the stools. Frequently when one does see a little blood in the stools it is thought to come from hemorrhoids and many patients with a malignant condition are treated for hemorrhoids because the physician does not want to soil his fingers by making a rectal examination. Even if a rectal examination is made the growth is often beyond the reach of the fingers and thus escapes observation. The question of proctoscopy does not come up, as a rule, until the condition is very far advanced. This woman just called in a physician a few days ago. When she came to me on the following day and was stripped and placed on the table I immediately felt the hard mass in the left side of the abdomen. In making an examination it is well to remember not to make the examination on a soft bed but rather on a table or hard couch. Proctoscopy was not done because the gravity of the condition was evident at once because of the bleeding and the tumor. I told the family that I could not tell where the growth started or what operative measures

might be indicated, but I advised them to give her to a surgeon and allow him to go as far as was necessary. These are the sad and hopeless cases that pass through the hands of the surgeon without receiving any help, and then go on to endure untold suffering. When a growth of this kind has metastases in the liver or the mesocolon or the mesentery an operation is only an added insult unless it is done for obstruction, and then we establish an artificial anus with the hope of mitigating the patient's suffering. It is like doing a gastroenterostomy for inoperable cancer of the stomach, where it is only indicated when there is an obstruction to the outflow of the stomach.

We will give this woman enough morphine to relieve her pain and allow her to go home as soon as possible, where she will be placed under the care of her family physician, who will give her whatever relief is possible.

*CASE III.—Malignant Disease in the Upper Abdomen.*—This is not a case to brag about. The patient is fifty-seven years of age and has complained of stomach trouble for two years, the chief symptoms being pain and vomiting. The family history is negative or at least has no bearing on the patient's present trouble. He has pain in the right side of the epigastrium one hour after eating, which is relieved by vomiting and this was considered an indication for a stomach operation ten months ago. Since this operation the patient has become weaker and weaker and vomits continually. There is a scar in the middle of the abdomen, but we have not been able to find out what operation was done. He has been brought down here to be reopened because sometimes even in a case like this something can be done at a second operation. There is a large mobile tumor the size of a child's head in the epigastric umbilical zone. Whether this tumor is retroperitoneal or not I cannot state. It has not the appearance of a carcinoma of the stomach. I have not examined this patient by rectum to-day. I am very much tempted to imitate Doctor Deaver, who says: "How is the diagnosis made? I am going to make the diagnosis with the aseptic knife." It is to be regretted that we cannot get the transcript of the operation performed on this patient at the other hospital.

We will make a right rectus incision. Examination shows no metastases in the pelvis. There is not the slightest indication of

any abnormality in the gall-bladder or in the liver, yet we have a growth that is a very large one, as large as a cocoanut or larger. There is an invasion of the mesentery and of the transverse mesocolon. The growth may possibly be a carcinoma of the stomach. There is a small growth in the omentum, and that is the only thing I find in the omentum. This does not appear to be a carcinoma, as the growth is not hard and there is an absence of other metastases in the immediate vicinity. You may recall that in malignancy in the upper part of the abdomen, and particularly in the stomach and first part of the intestine, one is very likely to find metastases in the cul-de-sac in the female and in the retro-vesicular pouch in the male. This kind of a metastasis when sufficiently large has been called a Blumer's shelf and is nothing more or less than a metastasis from a growth higher up. This metastasis is said to be due to a gravitation of the cancer cells from a higher region into this pouch. There they settle down and bud and after a time these budding cells produce nodules and we get nutmeg-grater-like effect and in time there is a confluence of these nodules along the back and across the pouch which forms a shelf. When the finger is inserted into the rectum it comes into contact with this shelf. Cases in which it is present are considered inoperable, as a rule, but in two instances I have done an operation on a stomach in patients where a Blumer's shelf was present. These operations were performed because there was an obstruction to the outlet of the stomach, also to give relief to the patient for a time. The patients recovered in both instances, one of them gained forty pounds and finally died of intercurrent cardiac disease. This patient had many metastases, three of fair size, one in the mesentery, one in the appendix and a Blumer's shelf. Hence it may be assumed that it does frequently pay to operate on cases even where there are a Blumer's shelf and nodules in the omentum. This patient, however, is inoperable—therefore we have simply explored his abdomen.

CASE IV.—*Removal of Enlarged Cervical Lymph-nodes.*—This patient is a girl, eight years of age, who gives a history of having had glands removed at the upper, outer and posterior angle of the jaw four years ago. These glands have certainly become enlarged very rapidly for they were noticed only one year ago. One is the size of an almond, at the upper, outer angle of the old scar. This

is a very beautiful scar. I would not object to being held responsible for the scar, but I should not wish to be held responsible for the glands that were left in at the former operation.

We will extend this transverse scar over the prominent part of the gland. I think we will find the glands broken down. I am correct in this; the glands are broken down. We will split the muscle longitudinally and get the capsule out if possible, because if we do not the wound will be longer in healing and it may be very discouraging. We will make a thorough operation, taking everything. There are a number of small glands just about to break down and possibly they are broken down in the centre. Usually an enlarged gland in this region has similar associates, probably four, five or six in number, and we wish to get them all out together with the capsule so that we will be more likely to get primary union. I am also going to take out the infiltrated muscle and put in a drain for forty-eight to seventy-two hours. We will use a rubber tissue drain and suture the edges of the sterno-cleido mastoid together to the emergence of the drain.

*CASE V.—Acute Appendicitis.*—This patient is a male, thirty-five years of age, admitted on April 27, complaining of pain in the right lower quadrant of two weeks' duration. His family history is irrelevant and has no bearing on his present condition. His health has always been good. He had the usual diseases of childhood, and typhoid fever at the age of fifteen years. For the past six years his appetite has been poor, digestion fair, no gas eructations or sense of fulness, and no jaundice. He has been constipated for the last year and has lost nineteen pounds in five months. He gives a history of having used alcoholic beverages for twenty years. His present illness began with an attack of sharp pain in the right lower quadrant and ever since he has had attacks of pain without nausea or vomiting. During the past two weeks the pain has been rather constant. Examination of the abdomen shows marked tenderness and some rigidity of the right side at McBurney's point. This man came near not being turned over to the surgeon. I was asked to operate on him when he had been here seven days. I said that as he had waited this long it would probably not hurt him to wait a few hours longer and make further progress in the control of his acute infection, as he seemed to be combatting it fairly well. He

had a white blood cell count of 10,200, red blood-cells 4,300,000 and 85 per cent. polynuclears. They made an exact analysis of the blood and faeces and the blood, creatin output, etc., and yet allowed this man to go on with his high leucocyte count without making a diagnosis of a surgical condition. On May 2 his blood count was 15,000 white blood corpuscles and 67 per cent. polymorphonuclears. I suppose they made an X-ray and catheterized him and yet they did not make the diagnosis. Finally when the man had had a temperature of 102° F., and a very tender mass in the abdomen for several days an operation was suggested.

This is a good time to call attention to how easy it is to "fall asleep at the switch." Every man does it sooner or later, and does it more than once. One wants to keep away from the man who never errs and from the surgeon who never has any mortality, for something is going to escape him and very badly when it happens.

I have now placed myself in a position where I must try to show that this man has appendicitis. The great trouble is that we depend too much on assistants and associates, when if we went into things ourselves and made a careful examination we would not allow these glaring facts to escape us. In this case there are two distinct things that may be troublesome. The first is the question of getting out the appendix and the second is the prolonged drainage that is necessary in such a case. If the appendix is removed within the first twenty-four or forty-eight hours in an acute attack the mortality is so small that it is not worth talking about, but in a case in which the inflammatory process has been going on as long as in this case it is a more serious matter to make a search for the appendix. Then there is the prolonged drainage to consider and the fact that one must make a longer incision than when the case is operated upon early.

You may recall that every now and then we used to have an appendix that we called extraperitoneal. The appendix is never extraperitoneal unless it is an anomaly. The so-called extraperitoneal appendix is an anatomical retroperitoneal appendix. The fear that we used to have of entering the peritoneal cavity no longer bothers us, for if we do enter the peritoneal cavity to-day it makes no difference if one is careful not to scatter pus. The pus in this case is of a dirty brown color and the appendix is densely adherent to

the iliac reflection of the peritoneum. It is a question whether we can do anything but just drain. There is a great deal of exudate. We will pack the cavity with hot sponges for a few minutes and then go ahead with the operation. If this operation had been done during the first two days of his attack we would not have had to make a four- or five-inch incision; we would not have run the risk of producing an intestinal fistula; and the convalescence would not have been prolonged. This patient will have to remain in the hospital from three to ten weeks, whereas if he had been operated on within the first few days he would have left the hospital in ten days. And that is only one item. We must think of the loss and expense that this prolonged convalescence means to the man and his family.

If I am unable to get at the appendix and do not think we should continue our manipulations any longer, I will establish drainage and close the wound. But I will follow the same procedure that I have for twenty-five years. I will go into the dense adhesions and free the serosa and musculosa of the appendix and expose the mucous channel, which I can strip out every time with a blunt instrument. This is a beautiful demonstration of how to get the rest of the stump. I first employed this method at the old Gouvernuer Street Hospital twenty-five years ago and I have followed it ever since. The appendix is sometimes hard to find. If we follow the white columns or bands which lead down on either side of the colon and cæcum we find they run together at the point of origin of the appendix. When the appendix has been traced to its origin the base is crushed, tied and cut off, smeared with phenol, and the cavity packed with iodoform gauze, etc. We pack the cavity with iodoform gauze as a means of haemostasis and also for drainage.

Now let us review our case. We have here a case of appendicitis, which has been on one of the services here for six or seven days. He came in with all the evidences of appendicitis that are recognized as such by the surgeon. The physician who had charge of him did not want him operated on, for what reason I do not know, for the patient gave a typical history of sharp pain in the right lower quadrant, fever, rigidity, and nausea and vomiting. There was every reason for operating on him early. An early operation would have forestalled peritonitis, prevented prolonged convalescence,

morbidity, and possibly future morbidity occasioned by adhesions, or possibly the appendix might have been left in the abdomen, due to inability on the operator's part in finding it, and therefore demand a second operation later.

There are instances in which delay in operating may be justifiable, as in an appendicitis complicating pregnancy, where there is no marked or aggressive symptomatology and in a case such as I had yesterday. In the latter case, a woman, fifty-four years of age, complained of loss of sensation in her fingers and progressive amnesia and aphasia. She was not in a condition to take an anæsthetic. A group of muscles in the arm and forearm were affected and though she had some extension of the fingers it was not what one would expect to find. Her condition relative to her appendix was as follows: The abdomen on the left side was soft; on the right side there was slight rigidity, and a distinct mass and tenderness were present over the area of the appendix. Her temperature was 104° F. and the pulse rate 100. In view of the progressive palsy, it was decided to postpone the operation until the brain condition cleared up and also due to the fact that the appendix was walled off and in a condition to be operated upon later without difficulty.

*CASE VI.—Herniotomy and Excision of Varicose Veins.*—This man is thirty-five years of age and has an enormous amount of varicose veins in the right popliteal space. He also has a hernia on the left side and a hernial scar on the right side.

We have observed, in many instances, that a patient with a distinct hernia on one side will have one on the other. This man was operated on five years ago for a hernia of the right side and he now has one on the left side. This hernia is due to heavy lifting and came on suddenly. It is of one month's duration. We will do the operation for the hernia on the left side and then for the varicose veins on the right side.

There is a bulging at the external abdominal ring on the left side. We will do the classical Bassini operation. The incision is made in a line from the spine of the pubes to the anterior superior spine of the ilium, that gives the point of attachment to Poupart's ligament. We start in at the external abdominal ring, making the incision about a finger's breadth above Poupart's ligament and long enough to cover the length of the inguinal canal. After going through

the skin and superficial fascia, the incision is carried down through the deeper tissues to the external oblique aponeurosis. We are careful to isolate the cord in the male and the round ligament in the female. We split the aponeurosis of the external oblique and then we strip off the cremaster muscle and push it to either side and bring the cord with its constituents up into the gap. After that we split the cremaster fascia, covering the cord, the size of which is reduced by cutting off the fat and superfluous tissue. In this case we will dissect well into the internal ring, free the sac from fat and tie the fat off. We will then reduce the stump beneath the arch of the internal ring and dissect out the sac, which shows very prettily. The gray membrane that you see is the hernial sac. The cord strips off readily, the vas showing up beautifully. We will strip the sac down well into the ring, and in the meantime we leave the cord on a retractor or held out of the way by a retractor. After seeing that there are no adhesions of the intestine or omentum we are then ready to clamp the sac and ligate its base. In this instance we will clamp and then cut off the redundant sac. Then we will transfix the base and tie with catgut. The next step is the repair of the inguinal canal. While the cord is held out of the way to one side by the retractors or tape, the conjoined tendon is sewed to Poupart's ligament by interrupted kangaroo sutures.

In this operation many men think they should be very careful of the nerve supply in making the incision to avoid injury to the ilio-hypogastric. There is a difference of opinion as to the result of cutting the musculo-cutaneous nerve. Doctor Moschcowitz says it makes no difference if one does cut the musculo-cutaneous nerve, while Doctor Dowd says that cutting this nerve results in a degeneration which has the effect of predisposing to a recurrence of the hernia. No one knows what the result of cutting the nerve really is except the clinician, who finds that even though he has cut the nerve no bad results follow. Now by sewing the aponeurosis of the external oblique together we close over the roof of the canal after having made the floor of the canal. We then close the wound without drainage. At about the ninth day we take out the stitches and allow the patient up and about. This patient will not be up so soon because we are going to do the operation for varicose veins.

*Varicose Veins.*—We will not enter into the causation of varicose

veins, though they have been attributed to increased cardiac pressure, changes in the blood-vessels due to infection, calcification, fatty degeneration, etc. We are concerned chiefly with the treatment. There are a number of methods of operating on veins. You may remember the method of circular incision, bisecting the veins and ligating them as they are bisected. Schede's method was to completely circumscribe the leg below the knee down to the deep fascias, thereby cutting across every vein involved and tie it on each side. Then came Fowler, who made a nick across a vein, then dissected it loose for a distance and then made another nick and brought the vein through this nick. There have been a number of modifications of the Fowler method and several different types of strippers have been devised, among them the Mayo stripper, by means of which the veins are exposed as in the Fowler operation and bisected and liberated, all adhesions and collateral branches being dissected away. Then we have the Trendelenberg method of removing four to six inches of the saphenous vein and deflecting the current so as to produce a deep collateral circulation.

In this case we will make an incision in the popliteal space about four inches in length. We find these veins in great clusters. I do not expect that we will be able to get all these veins out, but we will get the larger trunks. These veins are exceedingly brittle and one cannot do much with them. There are so many clusters and clots here that if we tried stripping these veins we would have trouble. We will make a second incision to get the other group of veins. There are certain elements of danger which we cannot eliminate, and these are thrombi and embolisms. There is always the possibility of an embolism getting into the circulation. Thus far we have records of three deaths from embolism in a very short period. The first patient I saw succumb to embolism after an operation for varicose veins was in the service of the late Dr. J. D. Bryant, in 1890. The patient was a personal friend of Doctor Bryant's and seemed to be doing very well after his operation, when he suddenly fell over and died. Doctor Bryant was greatly affected by this occurrence, more particularly so because he had assured the man that the operation was attended with very little danger, as it was a simple subcutaneous deligation not likely, so he thought, to be attended by embolism.

In operating and tying off the vessels we look for thrombosis as

a necessary step in the process of repair, but we do not look for embolisms that make an unfortunate permanent cure. Knowing this possibility we undertake an operation for varicose veins with more fear and trembling than we feel in doing a hysterectomy or an appendicectomy. This has been impressed upon me by three deaths from embolism within a year in the various services in this hospital. Such occurrences as this have made us a little careful and we do not allow the patients up after an operation for varicose veins until the tenth or twelfth day.

CASE VII.—*Excision of Cervical Lymph-nodes.*—This girl, fourteen years of age, has a large collection of enlarged glands in her neck on the left side. The posterior chain is involved, extending up underneath the left ear. They have gradually increased in size until within the last two weeks, when there seems to have been some reduction in the size of the glands. The patient has no pain and no evidence or history of tuberculosis or of tonsillitis. Occasionally we notice that after a tonsillitis the peritonsillar glands will become enlarged several weeks afterward. In such cases we find some other form of bacterial infection or we may find a tuberculous infection, and in some instances the contents of the glands have been reported to be sterile. Infection may also gain access to the glands through the mouth, from carious teeth or from the ingestion of bovine tubercle bacilli. It has been known to occur after scratches of the scalp or ulcerating disease of the scalp and even as a result of pediculosis capitis. When the enlarged glands are secondary to any of these conditions, there is no use in removing them until the source of the infection has been eliminated. Sometimes enlarged glands will subside after a tonsillectomy, but it is scarcely advisable to tell the family of the patient that a tonsillectomy will cure enlarged cervical glands, for you may not be able to fulfil that promise. We will make an oblique incision from the angle of the jaw to the clavicle. When glands are located as these are one must be very careful of adhesions to the jugular vein. I have seen them lay directly on the sheath of the jugular. We must also look out for the subclavian and the carotid arteries and for the pneumogastric and spinal accessory nerves. When dissecting in this region I have punctured the subclavian and sutured it up without any untoward results, but I am not advocating getting air into the veins. Injury to the spinal

accessory nerve may result in an uncomfortable paralysis, almost as uncomfortable to the surgeon who produced it as to the patient who got it.

In this case both the anterior and the posterior chains of glands are extensively involved and the sterno-cleido-mastoid does not amount to anything, being extremely small on the left side. The condition has more the appearance of Hodgkin's disease than of tuberculosis or a bacillary infection, and is not an acute process.

We have now dissected away the skin and superficial fascia and reached the chains of glands, clamping off the vessels as we have gone, and have come to where we see the spinal accessory nerve. We are going to have difficulty in keeping away from that nerve in this instance. We have had to cut the anterior half of the sterno-cleido-mastoid in order to save the spinal accessory nerve. It never does as much harm to cut the muscle as to cut a nerve. These glands lie right on the jugular. We have now exposed the descendans noni, which we will put on a retractor without making any traction, and I will hold it myself. Once in a while one's knowledge of anatomy is a hindrance rather than a good thing, as it may make one a little over-careful, but here we must be extremely cautious. We will now get the remainder of the posterior chain. To do this we will have to dissect them from the transversalis colli. A few of the glands are broken down. We will palpate the anterior chain before we stop. We use a No. 1 catgut for tying off all the vessels and we drain with rubber tissue.

The after-treatment in these cases is important. This case will be turned over to her doctor, who will see that she has proper hygienic surroundings, plenty of fresh air, nourishing food, etc.

**CASE VIII.—*Fibroid Uterus.***—Not all cases of uterine fibroid give symptoms. Where we find symptoms they are often cardiac in nature, such as dyspnoea, distress in breathing, or symptoms that are due to the fact that the fibroid is of such a size as to encroach on the abdominal cavity, causing pressure which interferes with the return circulation. Owing to the pressure on the bladder we may have the frequency of urination as one of the symptoms. In fact, many of the symptoms accompanying uterine fibroid are those resulting from the increased pressure.

In the presence of a fibroid of the uterus the question that comes

up is whether to do a myomectomy, a subtotal hysterectomy or a complete hysterectomy. Many fear to do a myomectomy because of the possibility of a subsequent malignancy. It has been shown that when malignancy occurs in the presence of a fibroid it always occurs in the uterus near the tumor but never in the tumor itself and that when malignancy does occur in conjunction with a fibroid it is frequently a sarcoma, four or five per cent. of fibroids showing this form of malignancy.

After having come to the decision that we are going to do an operation for fibroid, what are we going to do, a myomectomy, a subtotal or a complete hysterectomy? The sub-total hysterectomy means leaving only the cervix. In women under forty-five years of age we leave one or both ovaries so as to retain the mental balance. When a woman is forty-five years of age or over we know that it is only a short time until she reaches the menopause and by removing the ovaries we are not going to upset her mental balance so we produce an artificial menopause then and there. When do we do a complete hysterectomy and when a subtotal hysterectomy? In many cases in which the cervix is retained there is a persistent foul leucorrhœa. On the other hand, the removal of the cervix leaves a less stable pelvic floor, and hence in primipara or virgins we usually do a subtotal hysterectomy. It has been shown that malignancy is likely to occur in about 2 per cent. of the cases in which a subtotal hysterectomy has been performed.

This patient is thirty-eight years of age, a virgin, and has no leucorrhœa. In her case I am going to do a subtotal hysterectomy. I will make the incision in the median line, or practically so, and amply large to deliver the uterus without any difficulty. In doing this operation we always make a careful reflection of the peritoneum and are careful to avoid injury to the ureters. In this case we will leave one ovary at least. Having opened the abdomen we now dissect the bladder from the uterus. The next step is to clamp the broad ligament, both distally and proximally, and then cut close to the uterine vessels. We ligate all the vessels and cut the pedicle of the uterus at the cervix. We grasp the stump of the cervix with the Jacob's forceps and cut upward on the opposite side instead of downward, leaving the ovary on the left side. The tube is of no service so we will remove that with the uterus, as the tube if

left in might become obstructed and produce a hydro-salpinx. We are now ready for the haemostasis. We will make a transfixion with needle and gut, passing behind the uterine pedicle clamp and tie it off. I do most haemostasis work with a running suture. In an operation such as this there is exceptionally little danger of involving the ureter. I have stitched up the round and broad ligament and brought them over to the stump and I will stitch them to the side of the stump so that they will give additional pelvic support. Coming to the other side we tie off the uterine vessels and dispose of the round and broad ligaments as we did on the opposite side. We whip them over and when we come to the end we simply draw the suture up. The final step is the peritoneal toilet. We sew up the portion of the peritoneum which we pushed off the bladder zone, sewing well back and close to the cervix. We tie off all bleeding points before we drop the stump into the pelvis. There are at this point two things that we must look for, the appendix which has not been removed and the gall-bladder, which in gynaecological cases, and particularly in cases of fibroid of the uterus, is likely to be involved. I reported recently a series of three hundred cases of gall-bladder cases and in fifteen per cent. in which the uterus was examined there was a complicating fibroid. Later I reported three hundred cases of fibroids and a very large percentage of these were complicated with gall-bladder disease, and when we speak of gall-bladder disease we mean cholelithiasis. We will find the appendix and take it off, for many times an abdomen has had to be opened to get an appendix after a hysterectomy has been performed. The removal of the appendix is only a question of three to five minutes more, and it may save a very grave operation later on. We will now palpate the gall-bladder. There are no stones in the gall-bladder.

In this patient a myomectomy would not have been possible, owing to the general involvement of the body of the uterus in the fibroid condition.

The after treatment in this case will be one-sixth of a grain of morphine when the patient comes out of the anaesthetic and one-eighth of a grain when necessary for twenty-four hours after that time, and then no more unless it is absolutely demanded.

**CASE IX.—Thyroidectomy.**—This patient is twenty-eight years of age, and single. She came to me on September 6, 1917, with a

goitre, which gave no manifestation of either hyper- or hypo-thyroidism. She had been under the care of a physician, who gave her iodine. When first examined she had a pulse of 120, but a short time after she had been in my office it came down to 90. She had a mass on the left side toward the midportion of the neck, which looked very much as though it were cystic, but the growth may be hyperplastic or colloidal. I think, however, that it is a cystic or colloidal goitre. We thought that on admission to the hospital this patient's pulse would go up, but it was 100 and last night 99 and to-day 88. The reason she has come in to be operated on is because the goitre is beginning to press on the trachea and is causing her some difficulty in breathing, the breathing being stertorous at times; she also has some difficulty in swallowing. In cyst adenoma of the thyroid the treatment is excision of the cyst or cysts if multiple and in colloid thyroids we remove practically all of both lobes of the thyroid, if both are involved, without any hesitation whatever. Of course, when it comes to a hyperplastic thyroid, one may ligate very carefully one or both of the vessels and then later do a lobectomy, after preparation for the operation by the serum treatment or X-ray therapy. Personally I have no confidence in the X-ray, and in some cases it makes subsequent surgical work difficult, as it tends to cause fibrous adhesions. One thing that makes me think we have a cyst adenomatous condition of the thyroid in this case is that we have a peculiar symmetrical enlargement.

We will make a collar incision, beginning beyond the point of the sterno-cleido-mastoid in the left line and go around to the opposite side. We then dissect off the platysma and the superficial and deep layers and push the flap well up. I will make the incision one and one-half fingers above the sterno-mastoid notch. I clamp all bleeding vessels, as I prefer a fairly dry field, so that I can work more rapidly. Now we split down the median line and push the depressor of the thyroid to either side. The cyst is filled with a clear fluid and in dissecting it out we will have to be very careful not to incise the cyst wall. We will dissect it out with the gauze sponge. The cyst is substernal. We close the cavity with No. 1 catgut simply from the standpoint of haemostasis. We have to be very careful about tying off the superficial and external jugular branches, for if they begin to bleed we have a serious hemorrhage. We drain the wound from

twenty-four to forty-eight hours with a rubber tissue drain. Ordinarily the fluid in an adenomatous cyst is sanguinous or a chocolate brown, but this is rather peculiar; it looks just like water. I am stitching the platysma, the superficial and the deep muscles together to assist in the union and to make a better scar. We always like to make the skin line as delicate as possible, but there is no use of going into the detail of that as you are all familiar with it. The diagnosis in this case is cyst adenoma, requiring an extensive excision.

CASE X.—*Imperforate Hymen*.—We have here an unusual condition. I have seen only two similar cases in my twenty-seven years of practice. This girl is fourteen years of age and has an imperforate hymen. She came in with a history of having been unable to void her urine and was dribbling one-half to two ounces at a time. She was catheterized and two quarts obtained. There was a tumor mass, bulging at the vulva. It was thought that the tumor was caused by the menses which have been accumulating for months. The family history showed that two sisters have menstruated at the ages of twelve and thirteen respectively. The probability is, therefore, that the menstrual fluid in this case has been accumulating for two years, and had formed this enormous tumor which fills the entire pelvis. Within a year I have seen two cases of imperforate hymen and this is all I have seen in the twenty-seven years I have been associated with surgery and the twenty years in which I have done nothing but surgery. So look out for statistics. When a man says he has seen two of these cases within a year during which he has operated one thousand times you get quite a different idea of the frequency of these cases from that which you would get if you are told that he has met two of these cases in twenty-seven years and that for the past seven or eight years of that time he has operated from eight hundred and fifty to eleven hundred times a year. This serves very well as an illustration of how one may be misled by statistics, and of the necessity of regarding all statistics with suspicion. It is evident from my experience that imperforate hymen is a condition that does not occur very frequently. As to the question of what to do in a case of retained menses, it is better, as a rule, merely to puncture and to allow Nature to empty the blood as far as she can.

I am going to scrub this patient for operation myself so that

you can see the bulging of the vulva and the imperforate hymen, which are very evident. I think the girl had just been menstruating when she consulted me the other day and that accounts for the aggravation of the pressure symptoms. It is possible that some of the fluid is absorbed during the interim between menstrual periods and that this gives the patient some relief. The question here is whether to incise the hymen or burn it with the cautery. I intend simply to make an incision in this case. The incision, as you see, is followed by the evacuation of a large amount of dark retained menstrual fluid. We will insert a one-half or one-quarter inch rubber tube and leave it in to keep the incision open.

**CASE XI.—*Intrahepatic Stones.***—We have here a case that is the *bête noir* of the surgeon. This patient has been operated on twice before, and now she returns complaining of a recurrence of the same symptoms for which she was operated on before. She had a cholecystotomy and a choledochotomy and then a cholecystectomy and she now has a recurrence of her former symptoms. The patient is forty-five years of age and gives a history of pain in the right side. In the course of seven years she has been operated on twice for gall-stones. She first had an "ostomy" and then in January, 1916, an "ectomy." She had had both pain and jaundice since she had her "ectomy." The attacks come on around six p. m. or later and she has some nausea and jaundice. This is the second attack she has had within a week. The stools are acholic. She had a chill on January 16, 1918, but it has not been determined whether it is a nervous chill or not. There has been no loss of weight. Morphine must be administered with the attacks.

By abdominal examination nothing is to be found except the scar of the former operation. Her history is such that she is entitled to further operation. As I have said, these cases are the *bête noir* of the surgeon. The first man goes in and removes a handful or more of gall-stones and the symptoms return and the patient consults another surgeon. He operates and removes the gall-bladder, and says: "Now this patient can have no more gall-stones, for the gall-bladder has been removed." But he forgets the intrahepatic passages. The stone producer may be present in the small branches of the biliary system, due to the staphylococcus, or the streptococcus or the colon bacillus, etc., and this may give trouble even twenty-five years

after an operation has been performed and the gall-bladder removed. The first time I saw these intrahepatic stones was in a young girl fourteen years of age, and following the stones up I found that they came from the hepatic ducts and I did something in that case that I do not do now; I washed the liver. I injected salt solution into the hepatic duct and I do not know what it may have carried up into the duct, but a year later she had pain and at operation it was found that she had a stricture of the common duct. I then washed her out just as I had done the first time. That was the last time I washed out a liver. When we have an intrahepatic case there is no question that the stones may come down at successive times and one cannot guarantee to relieve the patient permanently by one operation, but in ninety-two to ninety-five per cent. of the cases there is no recurrence, while in the remaining five to eight per cent. we must expect to get a recurrence. This woman was operated on the second time by Doctor —, and certainly he did a thorough operation and left no stones. Understand that in this case two operations have been performed, and I have no anatomy to guide me, and I have the adhesions of the former operations to obstruct me. There is a great deal of oozing in the majority of these cases. I am first going to excise the old scar. I do not know whether the appendix was taken out or not. I always try at a secondary operation to get into the peritoneum either above or below the original cut and thus avoid adhesions. I am now breaking up old adhesions, and tying off blood-vessels. The liver has been exposed without much trouble. This woman is passing dirty bile and has a cholangitis. She may have a fibrous stricture or a kinking of the common duct. I have been asked if the stones I am finding are hard stones, the insinuation being that if they were soft stones they were recently formed and if they were hard stones they were left at the previous operation. The stones I am finding are hard. We will now travel along the right border and along the right lobe, breaking up the adhesions to what should be the position of the foramen of Winslow. I find a stone about the size of a walnut, and another almost as large. There are large numbers of small stones and a large stone in the common duct near the ampulla of Vater. We will dilate the duct and use suction and clean out both ducts. I think these stones were overlooked at the former operation.

I will introduce the index finger into the common and hepatic duct, and leading from the right hepatic to the left hepatic it is like a series of valves, resembling Burn's valves in the rectum. I go past the stricture point and find the duct strictured at the pancreas. We will now take a large uterine sound, though nothing shows one as much as the finger, and I am going to pass the sound through the ampulla of Vater into the intestine. It is a question where we have a distention like this and we have a small opening in front of it whether we will not find something in the dilated portion. We will put in a fair-sized tube. I am going to anchor the tube with catgut No. 2 or 3, and leave it in ten days for free drainage. The tube has gone through the common duct into the intestine. To be sure that the tube will be held in for a long time I will put in another catgut stitch. I am going to pack in around the tube iodoform gauze and put in a cigarette drain. I do not feel that these stones have been formed in two years; if they had been soft they might have been of recent formation. I cut out the scar in the beginning of the operation. It is said that in passing the needle through the dense tissue of an old scar there is a possibility of carrying infection into the wound. That is the argument, though it has never seemed plausible to me. In closing the wound we leave a liberal opening for drainage. I would ask you to refrain from tugging at the tube in the common duct until it loosens and comes out by light traction. I would like to have it remain in for two weeks at least.

When we speak of a constriction in the pancreas please remember the anatomy of the common duct. It may pass over or through the pancreas, or it may pass through near the surface of the pancreas. Remember that this is an anatomical and not a pathological stricture. I can pass a probe to the ampulla of Vater and sometimes the stones are in the sulcus. Many times I have gotten stones from below this peculiar constriction.

**CASE XII.—*Exploratory Laparotomy (appendectomy).***—The next case is an Italian, twenty-four years of age, who has been ill with stomach trouble for two years. He has lost twenty pounds in the last year. His history is rather indefinite, because he does not speak English. I understand that he does not vomit in the usual way, but that as he says he "throws off," or expectorates. According to his statement, he is distinctly better after eating. He has slight tender-

ness over the region of the appendix and in the epigastric zone. He was advised about two months ago that he ought to be operated upon. We will expose the appendix, making our incision sufficiently high to permit us to explore the duodenum, as his symptoms point to duodenal ulcer. The pain has sometimes been in the left lumbar region and he brings up a good deal of gas. It is said that at one time this patient's urine showed a large amount of albumen, but that I am inclined to question. The temperature is normal. I want here to call attention to the fact that gall-stones are more frequent in the female and duodenal ulcer in the male. In the female they are encountered most often between the ages of twenty-seven and forty years of age. Patients suffering from duodenal ulcer frequently show a change in temperament and frequently their true condition goes unrecognized for a long time, their trouble being diagnosed as neurasthenia, nervous indigestion, etc. Again many cases thought to be appendicitis have turned out at operation to be duodenal ulcer. Many cases in young women are diagnosed as appendicitis and at operation the appendix is found to be normal and it is discovered that the symptoms have been caused by cystic ovary; hence, in cases supposed to be appendicitis, in young women, it is well to examine the ovaries. Duodenal ulcer is usually relieved by operation, but the patient may return in six months or in one or two years because of a new ulcer or because the old ulcer is giving trouble or because there is an ulcer at the site of the anastomosis. It is unusual to lose twenty pounds in a year because of ulcer, unless the patient has been kept on a very rigid diet. The reason for not much loss of weight in duodenal ulcer is because the patient's distress is relieved by taking food and he frequently takes crackers and milk, ice-cream, soda water, etc., and this serves to keep up his nutrition. This man says he has lost thirty pounds and I question, if such is the case, if he has duodenal ulcer. It may be that he has an appendix situated high up posteriorly, or he may have kidney calculus. After meal distress never means kidney stones. We will make an incision that is high for the appendix and low for the gall-bladder exposure, and we will expose the gall-bladder and palpate it. I find no stones in the gall-bladder, but there are a few adhesions that do not amount to anything. The appendix and ileocecal valve are located high up, and possibly this is the cause

of all the trouble. The appendix is outside of its normal zone and turned up. We will clamp off the mesoappendix and tie off the appendix itself. We will then excise the appendix and invert the stump. When the appendix is sufficiently pathological it is very likely to be accompanied by pylorospasm, which may be productive of the symptoms this patient shows. Barring an exceptionally high swung duodenum I find nothing wrong in that locality. There is no duodenal ulcer. The duodenum is attached to the gall-bladder and swings well up above its normal zone. We will break up these adhesions. This patient has never given up entirely to the anæsthetic during the operation and the abdomen is not completely relaxed. In order to bring the wound together with entire satisfaction we will place a pillow under the patient's head, thus bringing the costal arch closer to the pubic bone, and thereby relaxing the rectus muscle enough to permit of a satisfactory apposition.

CASE XIII.—*Carcinoma of the Cæcum.*—This is a man fifty-five years of age, with a tumor in the left lower quadrant. He was admitted on the 29th of April, with a history of pain in the abdomen during the past three months. The family history has no bearing on the present condition. Tuberculosis and syphilis are denied. The patient had hemorrhoids removed in January of this year. The pain has been over the region of the stomach and toward the back. The pain is not influenced by anything, not even by lying down. Diarrheal attacks come on and he passes large quantities of dark watery stools. There is pain in the epigastrium no matter what he eats. He admits occasionally taking beer and whiskey. He has lost in strength and looks anæmic. There is a mass in the iliac fossa and tenderness. There has been a rise in temperature to 103 and then a fall to 101 2-5. To-day it is 101 2-5. The pulse has ranged from 100 to 80. His lungs are negative, and rectal examination is negative. The provisional diagnosis is carcinoma of the cæcum. The man looks like a case in which all we can hope for is an exploratory operation. The diagnosis lies between malignancy and diverticulitis. If he had had an acute diverticulitis for three months it would probably have perforated before this time. I have reported thirty cases of diverticulitis with gangrene or perforation occurring in the colon and cæcum. This man has diarrhea, and diarrhoea is not a symptom of diverticulitis, but a symptom of malignancy. All the older

text-books say that alternating diarrhoea and constipation are symptoms of malignancy. This patient has had two operations for hemorrhoids. While tenderness is not always present in cancer of the cæcum it is not a rare sign in that condition. Perforation in cancer is rare; in diverticulitis is not uncommon. It is exceptionally hard to get bowel symptoms in a growth in the cæcum and colon, and unless the stools are carefully watched for blood or a proctoscopic examination made it will escape attention until it is far enough advanced to cause pain or give symptoms of obstruction. We will make a right rectus incision and then proceed to make an exploration of the abdominal contents. The right lobe of the liver is free from any deposits whatever, also the left. There is perforation of some sort for some reason, involving the cæcum behind and well into the front. The omentum is wrapped around the site of the infection, just as it does in the appendicular cases. There is a growth, which is inoperable. If the man will stand for it we will put him on a Coolidge tube treatment and give him several sittings. He has not been obstructed, and hence an artificial anus is not indicated at the present time. We will pack with iodoform gauze and will leave the wound wide open, and we can then give him three, four or five treatments with the Coolidge tube.

**SURGICAL CLINIC**  
**OF HUGH N. MacKECHNIE, A.B., M.D., C.M.**  
**AT LOYOLA UNIVERSITY MEDICAL SCHOOL. CLINICAL CONGRESS OF SURGEONS**  
**Chicago**

---

**CHRONIC CHOLECYSTITIS**

**CASE I (G. H.).**—This patient, a man aged twenty-three, came to me June 8, 1917, complaining of pain in his right side—sometimes in the hypochondriac region, sometimes in the lumbar, extending downward and to the front into the right thigh—sometimes, and most frequently, only in the right iliac region. It became quite severe when he would sit or stoop over at his work for a considerable period. Most of the time he had a dull aching pain, but rather acute attacks would come on at various times. He was troubled a great deal with a marked amount of intestinal fermentation. He was becoming quite constipated, with dry, hard stools of normal color. Had never noticed gray, black, or red stools. After attacks he sometimes had frequent liquid, hot burning stools for a day. At times, when attacks were on, patient would urinate frequently, especially at night. He had never noticed any blood nor marked dark sediment in the urine, but frequently had seen a light flocculent sediment.

These attacks came on without warning, with little or no nausea and frequently with vomiting—emptying the stomach of most of its contents with one or two attempts. Patient had never been jaundiced; skin was frequently muddy in appearance. Tongue was coated most of the time, and particularly during the attacks.

*Examination.*—Patient fairly well nourished; short and thick set; eyes are set well in, with dark circles beneath; sclera not clear; the skin is dirty in appearance. No evidence of jaundice. Tongue thickly coated white, dry, and tremulous.

*Temperature.*—99.4° F. Pulse 84.

*Blood Examination.*—Whites, 11,500; differential normal; polymorphonuclear, 85 per cent. X-ray, as you see, gives no evidence of disturbance in the kidney, ureter, or gall-bladder. No barium meal and fluoroscopy made because of lack of indication.

*Urine.*—Negative for any indications of renal trouble.

*Thorax.*—Negative.

*Abdomen.*—Slightly increased resistance in right rectus. Stomach slightly enlarged; general visceroptosis. Some tenderness in epigastrium just above the umbilicus, also to the right of it on deep palpation. Subhepatic palpitation and hammer-stroke percussion did not elicit much reaction. On deep pressure marked tenderness over McBurney's point. Kidney on right side palpable with difficulty—not enlarged nor tender. Left kidney the same. No tumor mass or other sensitive spot found.

The diagnosis must take into consideration cholecystitis, renal lesion—calculous or tuberculous—and appendicitis. Cholecystitis was indicated by the pain in the right hypochondriac region, referred through to the back, the tenderness opposite the tenth costal cartilage, and the chronic indigestion. There was, however, no evidence of pathology to produce the blood-picture which we found. The age and the regular habits of the patient, the lack of typhoid or septic infection, and the absence of a primary acute attack contra-indicated gall-bladder trouble.

Renal lesions, both calculi and tuberculosis, were eliminated by the examination, the negative urinary and X-ray findings.

The appendix was sensitive and the right rectus was resistant; the pain with nausea, together with slight fever, indigestion, and constipation, indicated an appendicitis.

Diagnosis then was subacute appendicitis and a possible chronic cholecystitis.

*Operation.*—The primary incision was made along the border of the right rectus and the appendix found and removed. It was long and club shaped, with several points of fibrosis along it. The end was quite enlarged and full of fecal matter. It was removed. The fingers were inserted up to the gall-bladder. This organ was found enlarged, not capable of being emptied, and containing many stones. Through a puncture wound down onto my finger I drew up the gall-bladder, opened it, removed the stones, and inserted a rubber-tube drainage. There was no evidence of pus, but a thick black bile was found. No calculi were found in the ducts. The appearance of the mucous membrane did not indicate the necessity of removal of the gall-bladder.

Recovery was good and the patient left the hospital in two weeks. The gall-bladder drained for five weeks, during which time it closed three times, but opened again. This is quite the usual procedure where we have an infection that has not been thoroughly eliminated.

I advised an early excision of the viscus, which was refused.

He returned to work four different times, and each time had a mild return of symptoms and is now ready to accept my advice. In these attacks the pain is located over the gall-bladder, and deep palpation the patient resents very markedly. Palpation over McBurney's point does not produce pain.

*Operation.*—The incision is made around the old scar and down through the peritoneum. These omental adhesions we cut and tie off. This was Nature's first method of cofferdamming around our gauze drain, and she did it well, for there was no leakage with peritonitis at any distance from the tube.

The gall-bladder is isolated, and is found quite long and full of bile and mucus. The ducts appear free and patent and contain no stones.

The next step is to clamp the cystic vessels and duct. This we do by running a thumb and finger down to the foramen of Winslow, locating the common duct, following it down to its entrance into the duodenum, then returning and following the hepatic up to the fissure of the liver. Again we return to the junction of the cystic and hepatic ducts, retract onto the cystic just far enough to get space to apply an eight-inch forceps. Then distal a second forceps is applied. One may tie the duct and cystic artery separately, but this adds a difficulty, especially in fat women, is not essential to safety, and therefore is of doubtful value. I believe that in those cases where hemorrhage occurs it is not due to the inclusion of the duct and artery, but to a lack of fixation or proper tying of the suture.

The duct is now cut between the forceps; the peritoneum is reflected from two-thirds of the surface of the gall-bladder and the viscus separated from the liver by careful blunt dissection and removed. The peritoneal edges are sutured over and against the raw bed on the under surface of the liver. Sometimes I sterilize the cut end of the duct with phenol and alcohol. A cigarette drain is put in down to the end of the duct, and at times a second one to

the renal fossa. The wound is closed with catgut and silkworm and horse-hair up to the drainage.

This gall-bladder, you will note, has a marked fibrosis around the point where the primary drainage was inserted; that it had some mucus and bile in its lumen; that its walls are thickened, and I may say its size is much less than at the previous operation, as is characteristic of chronic cholecystic cases; and that its mucosa is markedly hyperæmic.

This case raises the question, When shall we and when shall we not remove the gall-bladder? On one hand we have those who hold that a prolonged drainage is sufficient to remove the infection, and that excision is too severe an operation to serve our purpose. On the other hand, we have those who maintain that a prolonged drainage does not always remove the infection, as evidenced by recurrences; that the gall-bladder has no function in the human economy, and that the operation is not such a dangerously severe one; therefore it should always be removed.

Infections gain access to the gall-bladder (1) by passing up the ducts from the intestine; (2) by contiguity of tissue, as when an ulcerating intestine and a necrotic gall-bladder are touching; and (3) through the circulation, either lymphatic or blood. The first leads most frequently to an infection of the contents of the viscus, with the development of an empyema and a later infection of the walls. The latter two lead to an infection primarily of the walls and, later, possibly to an infection of the contents. The former probably will recover with drainage if it has not been allowed to run to the second stage. The latter will undoubtedly do better with—and I believe requires—an excision as the only means of getting rid of the infection. There are contra-indications for excision, but I believe they maintain as much for one as for the other, and that almost every case that will stand a drainage operation will stand a removal.

I am therefore in favor of, and do practise, excision in those cases with gall-bladder infections, either primary or secondary to stones, that can stand a major operation, with the infection in the gall-bladder wall, as the only means of getting rid of the infection; and those cases of empyema where the gall-bladder can be removed without rupture and infection of surrounding parts.

## APPENDICITIS AND PERITONITIS

CASE II (R. M.).—This young boy, aged seven, has been complaining for three days of stomach-ache. He began with a slight umbilical pain and a little nausea for a short time. The pain persisted all evening, but was relieved at night by an enema when he passed some gas with hard, dry faeces. His bowels had been regular, but had not moved that day. He appeared to have some fever and was restless all night. In the morning he had slight pain, but ate a good breakfast and went to school. During the day the pain grew worse, and when he got home from school it was quite severe. He now said it was all on the left side. His parents thought the pain was due to his being thrown into water at noon and sitting with wet clothes in school. About 2 A.M. the pains grew much worse, and in the morning they called in a doctor, who diagnosed acute appendicitis with peritonitis. He was brought to the hospital, and we find, on examination, a well-nourished boy with good color, highly flushed, with a pinched expression, tongue coated white and dry; lying on his back with knees drawn up, abdomen tympanic, but very little distended and firm, with more resistance on the left side, but on deep palpation the pain appears on the right. There is some dulness in the flanks. Temperature, 100.4° F.; pulse, 120; respiration, 26. White blood count, 18,500; polymorphonuclears, 90 per cent.

We have here a case of evident peritonitis following a rupture of some viscus, either an appendix, appendix epiploica, or the intestine or stomach. It is not ileus, because of the history of short duration, the mildness of the symptoms of obstruction, the type of distention, and development of peritonitis.

The pain on the left side with nausea in the early hours, the temperature, pulse-rate, and the persistence of the left-sided pain make us think of an inflamed appendix epiploica, but deep palpation gives more resistance in the right than in the left rectus. We have no history of bowel trouble to make us suspect ulceration; no diarrhoea nor chronic constipation; no blood in the stools.

On the other hand, we do have a history of pain with nausea, coming on rather acutely and increasing in severity; of relief from pain supposedly due to relief of intra-abdominal tension following an enema; of an increase of fever with a seeming improvement in

his condition; with a high leucocyte count of the polymorphonuclear type, coincident with his fever and with the development of tympany and free fluid in the abdomen. This is the type of history we get with an acute gangrenous appendicitis, with rupture and the development of peritonitis.

Now that he is partially asleep, we palpate the abdomen, and we find that there is a noticeable resistance in the left rectus, but that it is much more pronounced in the right. We feel certain now that our diagnosis is correct, and we make our incision at the border of the right rectus muscle. The peritoneum is markedly hyperæmic and thickened as in peritonitis; on opening we find a belly full of seropurulent fluid with some flocculent material. I insert my finger along some thin fibrinous adhesions beside the cæcum and readily find the appendix, thickened and firm and bent upon itself. It is brought into the wound and removed. I now irrigate the whole abdomen with normal saline solution, beginning at the left renal region, then to the right renal, the left iliac and pelvic, and, lastly, the right iliac. I do this because I believe it is good treatment to get rid of as much septic material as possible, even at the risk of removing antibodies and much defensive material; feeling certain that Nature can better sacrifice a little of the latter when she has so much less of the former to fight. My results justify my action in the matter. I would not consider doing this in cases where the infection were walled off or where much traumatism would be done, thus opening up fresh avenues for absorption which had been already closed.

Non-irrigation has the advantage of not disturbing adhesions, of not opening up closed avenues of absorption, of not traumatizing tissues, and of not washing away the antibodies developed to take care of the existing infection. Irrigation has the advantage of removing a large quantity of septic material, of producing a large flow of blood to the abdomen and with it an increase of antibodies; it dilutes the toxins being absorbed, and it always decreases the dreaded ileus of those cases. On the other hand, it should not break down adhesions, nor traumatize tissues, nor carry infection to parts where it has not already reached. In many of these cases of the less virulent and less extensive type I frequently put two or three ounces of ether in the peritoneal cavity and close without drainage. The

results are most gratifying. The patient sleeps much longer than after the ordinary anaesthetic, wakens quietly, and suffers less early pain and less ileus. It would require much temerity to use it in so pronounced a case as this.

We now insert two cigarette drains—one leading down to the pelvis and the other to the edge of the cæcum, just below the ileum. The abdomen is closed up to the drains with through-and-through silkworm gut and a few interrupted coaptation catgut sutures in the peritoneum. He will now be put to bed in the semi-Fowler position, lying on his right side. This position favors drainage from the sub-hepatic region and also from the left side to the most dependent or right side, where the drains are. He will be given proctoclysis as tolerated.

When shall we remove the appendix accompanied by peritonitis and when shall we merely drain, getting the appendix at a later operation, provided it gives trouble?

Conservatism has been the watchword in these cases, tending to leave the appendix and trusting to Nature to slough off the offending organ during drainage, or to produce a sufficient fibrosis that the organ would be obliterated, or, if neither occurred, that a subsequent attack of appendicitis would never take place. I believe we have made many errors in the past and that we ought to remove every appendix that we possibly can get. Those that are walled off in an abscess, or that can be gotten only by breaking down extensive adhesions, or that necessitate much traumatism, or in which the patient can not stand the extra shock, should be left, even with the possibility of a second operation.

The case with the small intra-appendicular abscess which is readily enucleated without danger of rupture, or the acute appendix that can be separated without breaking down any limiting plastic wall, or the one with the general peritonitis and a belly full of pus or seropurulent material in which the appendix can be elevated, should be removed at the primary operation. In these cases Nature has put forth her best efforts: she has filled the abdomen, either locally or broadly, with defensive material, and if the primary focus is removed and further offence be not given she will be able to take care of the trouble caused. On the other hand, if this offending

organ be left in to keep up an offensive, Nature may not be able to keep up a sufficiently prolonged fight and may have to surrender.

This appendix, as you see, has a large gangrenous area with perforations near its distal end, in accordance with the history. Nature has made most strenuous efforts to wall this off, as evidenced by fibrinous plastic material adherent to and around it. Unfortunately the amount of material thrown out through the perforation was scattered too broadly and too quickly and a generalized instead of a localized peritonitis was produced.

#### BIRTH-PALSY

**CASE III (P. L.).**—This boy complains of an inability to raise his right arm to more than an angle of 45 degrees from the body; that he can not bring the arm across the body; that he cannot flex the forearm less than a right angle; that his forearm is in continued pronation and that he has little ability to supinate, and that the right arm is much smaller than the left.

He was a breech-presentation case and is supposed to have had a fracture of the upper arm or clavicle at birth, which is held responsible for the present condition. He is a bright, active boy, well nourished and well developed.

The right shoulder and arm are not so well developed as the left. The right deltoid circumference is three-fourths inch less than the left, and the right biceps one-half inch less than the left. The forearms are of equal size; the right humerus is one inch shorter than the left. The radius and ulna are equally long on both sides; the right supraspinatus is smaller. The right shoulder-joint, clavicle, and scapula are elevated three-fourths inch. He has good power in the extensors and flexors of the wrist and hand, but does not close the right hand as tightly as the left. Wrist motion is good. Reflexes, both superficial and deep, are not altered.

The skiagraph shows the right shoulder slightly raised, with a little variation from normal in the relation. The humerus is small and the head poorly developed and placed higher than normal in its relation to the other bones. It moves rather freely in the glenoid cavity, but appears small and is not normally prominent in the axilla. The radius and ulna are normal. The elbow-joint is normal.

It is evident that at no time was there a fracture either of the

humerus or the shoulder girdle. There is no deformity of the bones with such pressure on the nerves as would produce this condition. The paralysis of poliomyelitis succeeding an attack of fever, with more or less definite pains, is not usually so evident so early in life, and is not confined so strictly to a definite group of muscles. It may be progressive in its development and it may be progressive in its improvement.

Spastic cerebral paralysis is usually ushered in with one or more convulsions, although its onset may quite resemble that of poliomyelitis with fever, restlessness, etc. Early paralysis occurs of a hemiplegic or diplegic type, usually, and never of a small group of muscles. In this type, however, there are always exaggerated reflexes, some rigidity, and electrical reactions.

The early development of this case, the localized distribution of the trouble, the extent of the paralysis, the lack of spasticity, and the normal reflexes point to one of birth-palsy. Of this condition we have two types—the upper arm and the lower arm.

If you will recall the anatomy of the brachial plexus you will remember that it is formed from the anterior roots of the fifth, sixth, seventh, and eighth cervical and first dorsal. The fifth and sixth unite to form the upper cord, the seventh forms the middle, and the eighth and first dorsal form the lower cord. Each of these cords divides into an anterior and a posterior branch. The three posterior branches unite to form the posterior trunk; the anterior branches from the fifth and sixth with the seventh form the outer, and those from the lower form the inner trunk.

The muscles of the upper-arm type of paralysis, viz., supraspinatus, infraspinatus, rhomboids, servatus magnus, pectoralis major, biceps, coracobrachialis, brachialis anticus, brachioradialis, subscapular and teres major, deltoid and teres minor, are supplied mostly by the fifth, to a less extent by the sixth, and to a small extent by the seventh.

Those of the lower-arm type, viz., triceps, flexors, and extensors of the wrists and hands, intrinsics of the hand and supinator brevis, are supplied largely by the eighth cervical and first dorsal, less by the seventh cervical, and a small part by the sixth cervical. It will be seen that the fifth cervical is the most important root in the upper group, and that the eighth is the most important in the lower,

with neighboring nerves adding their quota in much smaller amounts. It follows that injury to the fifth and the eighth would produce the greater degree of paralysis in their respective groups, and that supplementary injury to neighboring nerves adds to the degree of paralysis. On the other hand, a lack of involvement of these other nerves will decrease the amount of paralysis. This is the case with this patient. We have here a partial paralysis of the upper-arm group of muscles, but have some action in almost every one of the group. This would indicate to us that the main nerve, the fifth, is involved, and that probably the sixth and seventh are less so or not at all. This bears out the work of Sever in his researches on cadavers of infants.

Of the etiology of the condition: Pressure by forceps or finger during delivery, by fractured clavicle, hemorrhage into nerve, tearing of nerve by pulling, pulling on neck with head or with shoulder impacted, twisting and turning. These all had their proponents. There is undoubtedly a hemorrhagic infiltration, a rupture or tearing of the nerve-fibres or sheaths. It is possible that in a few cases the trouble may be high up in the nerve-root or even in the cord itself. Sever, however, has shown that tension on the head with the aftercoming caught shoulder, or *vice versa*, produces tension first on the fifth root, and with continued pulling tension on the sixth and next on the seventh. In this manner, if any damage is done, the fifth suffers first, and if the force persists it may be extended to the sixth and seventh. Not only does the tension from pulling, but the twisting and turning incident to the effort to release the head or shoulder markedly tend to increase the trauma.

Unfortunately for these cases, it is some time before it is recognized that any damage has been done to them. The lack of coördinate movements is not appreciable, because of the ignorance in such matters of its parents, and not until the disparity in activity between the two sides is seen is the physician summoned. By this time the fibres have so retracted, the exudate has become well organized and reaction of degeneration has occurred, and the period of best possible results has passed. If they could be seen at this stage and were able to stand such an operation we should get at least a fair degree of repair and results.

In the very early stages massage or mild manipulation tends to

stimulate repair, and if too much damage to surrounding structures has not occurred we may get good functional results. If, however, fibrosis and retraction of muscles and fascia has taken place, this treatment does not avail and surgical intervention is necessary.

If these cases come to us late we must consider the intrinsic nerve injury and destruction, the muscle contraction and fibrosis, and the bony deformity before operating or giving our prognosis to our patients. Taking these points, then, in this case, we do not feel that we can give this boy or his parents a sufficiently good prognosis to warrant an operation on him.

## THE NON-SURGICAL TREATMENT OF ENLARGED PROSTATE

By G. FRANK LYDSTON, M.D.

Formerly Professor of Genito-Urinary Surgery and Syphilology, Medical  
Department State University of Illinois, Chicago

---

I BELIEVE that the specific for prostatic hypertrophy is the knife. Anything else, in the present state of scientific knowledge, is a compromise. I nevertheless desire to protest against the immediate and indiscriminate performance of prostatectomy in every individual past middle life in whom the prostate is found to be enlarged. There are cases in which enlargement is discovered early where compromise is the better part of wisdom, and later cases where compromise is unavoidable. Not always is it an easy matter in subjects past middle life to decide where inflammatory enlargement ends and true hyperplasia or hypertrophy begins, especially if the practitioner is not expert with the cystoscope nor, indeed, always when he is expert. Neither is it always easy in a case presenting itself for examination for the first time to determine to what extent the symptoms are due to permanent obstruction and to what extent to superadded inflammatory conditions, which, perhaps, in future may be avoided.

When employed early, measures of palliation, such as proper hygiene, hydrotherapy and massage, not infrequently are curative to a certain degree, by reducing inflammatory complications and preventing the development of sufficient permanent prostatic change to produce obstruction. This naturally follows, if there is anything in the view that chronic inflammation is a factor in the etiology of prostatic hypertrophy. It must be remembered that: 1. A moderate hypertrophy may produce symptoms because of more or less recent inflammation. 2. Elderly subjects not infrequently present themselves with infective—often specific—prostatic inflammation more or less recently acquired, perhaps superadded to a slight or moderate enlargement or to a chronic, purely inflammatory process. Judicious

massage and auto-vaccines often are successful in reducing the size of the prostate and preventing further hyperplasia in the first class of cases and in practically curing the second. It is not always wise to immediately operate cases in which the symptoms are comparatively slight and readily relieved. It is not rare that the surgeon advises operation in cases which remain comfortable for years after operation is declined. Sometimes they apparently recover altogether from their prostatic symptoms. Many patients refuse operation under all circumstances. The conscientious surgeon always informs the elderly patient who is suffering from prostatic hypertrophy that the condition generally is progressive, but in some instances, the patient refuses to submit to the advised operation so long as suffering is not acute and the sexual power remains unimpaired.

Patients of from 50 to 65 years of age who are in full sexual vigor and are not yet greatly discommoded by their prostatic disease, are quite likely to demand a guarantee that a prostatectomy will leave their sexual function unimpaired. While, under modern methods, the operation does not often destroy the sexual power, unless it be comparatively feeble before the operation is performed, the conscientious surgeon naturally declines to give the positive assurance of safety demanded by the patient. Under such circumstances it is not surprising that operation so frequently is refused, or at least deferred either until severe symptoms develop, or the sexual power becomes so feeble that the patient is indifferent to the results in that particular respect. Our duty in such cases is plain, and the management of the case essentially is what the surgeon, were he similarly afflicted, would be likely to choose for himself.

A symptomatic enlargement of the prostate never justifies prostatectomy. Some such cases go through life without a symptom.

It thus will be seen that there are cases in which the surgeon conscientiously can keep the patient under observation and treatment pending the absolute necessity of prostatectomy, or while waiting for the patient's consent after the necessity of prostatectomy has been proved. Much often can be done for the patient under such circumstances.

The results of prostatectomy are so good, even in advanced life, when performed before serious infection of the bladder or infective-obstructive derangement of the kidneys has occurred, that it often

is safe to defer operation for a time, always providing, however, that the patient is under constant supervision and within reach of expert assistance. The palliative measures already suggested, and especially systematic massage—by removing inflammatory "plus" conditions—often are productive of excellent results. Indeed, when the cases present themselves early, it by no means infrequently happens that operation can be indefinitely deferred, a symptomatic cure resulting and the prostate becoming markedly reduced in size, which is as much as can be asked under the most favorable conditions.

The author is convinced that many prostatectomies have been done by non-expert routinists for prostatic enlargement in men past or even below middle life in which the condition was largely, if not altogether, one of chronic inflammation, remediable without prostatectomy. That prostatectomies have been done on quite young men with gonorrhœal prostates, is one of the many blots on the history of surgery. It is the author's view that most of these cases would be better for incision and drainage, but prostatectomy is rather too radical.

Accepting the view that hypertrophy of the prostate often is due to chronic inflammation of some variety, it by no means is astonishing that good results sometimes are obtained in early cases by non-surgical measures of treatment. The employment of auto-vaccines in mild, or even serious, infections of the prostate and bladder in comparatively early cases of prostatic enlargement is directly in line with what we know regarding the rôle of infection—especially by the *bacillus coli*—in prostatic hypertrophy. Even in moderately advanced cases with superadded infection, where operation for any reason is impracticable, auto-vaccines may be of great service.

When once an attack of retention has occurred, it usually is not wise to institute non-surgical measures of treatment, save under the stress of absolute necessity and then only under vigorous protest. One attack of retention almost inevitably means another, sooner or later, followed by a succession of attacks and, unless operated, finally by catheter life. Once catheter life has begun, the end usually is in sight, the rare exceptions merely proving the rule that, on the average, death—and a by no means pleasant death—occurs within five years.

Although by no means a therapeutic optimist with regard to the

medical treatment of prostatic hypertrophy, the author is convinced that the administration of thiosinamin is very useful in some cases. It doubtless has no value in reducing adenoma of the organ, but it sometimes really does seem to be of great service where there is a complicating inflammatory condition and in preventing, or possibly reducing, fibrotic changes, or even inhibiting further adenomatous development. In early cases, therefore, and where for any reason prostatectomy is inadvisable or impracticable, the drug is well worth a trial.

Electricity has been extolled for the cure of prostatic hypertrophy, and has been the *pons asinorum* across which multitudinous quacks have travelled to prosperity. Electricity, however, never cured a case of true hypertrophy. The alleged "cures" have been either cases of chronic inflammation which sometimes is benefited by the galvanic current, or cases in which no prostatic disease of any kind existed.

According to the author's observations, electricity in prostatic hypertrophy is not only useless, but more often harmful than otherwise. He has met with numerous instances in which serious injury resulted from its use. One instance within his knowledge was especially distressing in that the victim was an old and valued medical friend. This gentleman had decided to submit to prostatectomy, but subsequently permitted himself to be sidetracked into the hands of a non-operating "electrologist." The result was a suppuration of the prostate from which, after months of suffering, the patient finally was relieved by operation. The prolonged suppuration and pain, together with the pyo-toxæmia and a low grade of pyelitis, however, had produced a condition of chronic invalidism from which the patient never recovered. He died about two years later from uræmia following an acute exacerbation of pyelonephritis. And the electrologist is sometimes in good surgical company; in another case, related to me by a professional friend, a man of 60, with slight prostatic symptoms, no retention, moderate enlargement and no residuum, was rushed to the operating table. Death resulted from secondary hemorrhage.

Speaking in a general way of the non-surgical treatment of prostatic disease, and thoroughly in accord with the view that, broadly speaking, the treatment of hypertrophy of the prostate now is a

strictly surgical problem, the author nevertheless hopes that some method of treatment eventually will be developed which, under favorable conditions, will enable the profession to dispense with the knife in at least a reasonable proportion of cases. When once the profession and the laity have learned the lesson of prostatic hygiene, we may have the opportunity of treating cases in their incipiency and the non-surgical treatment of prostatic hypertrophy may become a very important field of medical endeavor.

As illustrative of the fact that conservative treatment sometimes is followed by excellent results in symptomatically early cases of prostatic enlargement, the following cases of the author's are suggestive:

**CASE I.**—A man, aged 52, with a history of an uncomplicated gonorrhœa at 20, who later had noticed prostatic symptoms for several years, experienced great difficulty in micturition following exposure to cold and wet, associated with dietetic and sexual excesses. Temporary retention came on, but was relieved by hot baths without instrumentation. Examination showed a greatly enlarged, somewhat tender prostate, and an infection of the bladder that proved to be bacillus coli. There was no temperature and no symptoms pointing toward abscess. The patient was informed that he probably sooner or later would require a radical operation, to which he replied that he "might require it," but never would "submit" to it. This case has been under observation for fifteen years. Under massage and proper hygiene the prostate diminished considerably in size and there never has been another attack of retention. The colon bacillus infection never has cleared up entirely, but latterly has been immensely improved by auto-vaccines. The prostate now is only moderately enlarged and under occasional massage apparently is giving rise to no trouble.

Another very instructive case is the following:

**CASE II.**—Man, aged 63, physician. Obscure history of gonorrhœal infection during his "college days." Did not recall whether or not he had any complications following the gonorrhœa. For several years the patient had been rising at night and the urinary stream had been "slow." Never had any symptoms of stricture. The day before the author saw the case, the patient had been exposed to the severe spring weather then prevailing. Acute reten-

tion developed and relief was sought. A uniformly enlarged and moderately tender prostate was found. Later examination of the prostatic and urethral secretions showed no gonococci. No other organisms were sought for. The catheter was necessary for several days, after which time micturition became normal. Operation was advised and refused. Under massage and hot rectal irrigations with hygienic regulation for about three months, the prostate became considerably smaller and the stream freer. The case now has been under observation for seventeen years, during which time an occasional massage has been given. The prostate still is distinctly enlarged, but has caused no particular trouble since the single attack of retention many years ago, and the patient, now 80 years of age, naturally does not take kindly to the idea of an operation.

In cases like the foregoing the inflammatory and infective elements are the essential features, it is true, but if the view that infection and inflammation are important etiologic factors in true hypertrophy is correct, then we are right in regarding them as moderately developed cases of that disease. It hardly is reasonable to suppose that, if proper treatment had not been instituted, the patients would not later have suffered from serious obstruction necessitating a prostatectomy. The foregoing cases merely are typic of a considerable number which have come under the author's observation, and have been recounted simply to emphasize the fact that an increase in the size of the prostate in men at or past middle life is, when taken alone, not necessarily an absolute indication for a radical operation. This much is certain, viz.: similar cases—and even cases much less important—are being operated on daily.

One of the principal dangers of conservatism in prostatic disturbances is overlooking early development of malignant disease. Carcinoma may not cause subjective symptoms productive of much discomfort until quite late in the course of the disease, hence it is in just such cases that conservatism is likely to look most promising and for a time may appear to be very successful. Nodular and indurated prostates after middle life are to be regarded with suspicion and, other things being equal, demand immediate removal, even though the subjective symptoms are a negligible quantity.

# Medicine

---

## A CASE OF ACROMEGALY OF APPARENTLY ACUTE ONSET

By S. A. LOEWENBERG

Philadelphia

Associate in Clinical Medicine, Jefferson Medical College. Assistant Visiting Physician, Jefferson Hospital, Visiting Physician, Philadelphia Hospital and Eagleville Sanatorium for Consumptives

---

WITHIN a decade and a half, after Marie in 1886 first described two typical cases of acromegaly, many such cases, either typical or atypical, have appeared in literature; and one feels that an apology is almost due for thrusting one more case upon the reading profession. However, since this case presents several interesting features, I feel justified in offering it.

Most authors attribute acromegaly to disease of the pituitary body. Dean D. Lewis<sup>1</sup> believes with Bendas that acromegaly is caused by hyperplasia of the chromophile cells of the hypophysis. Rolleston<sup>2</sup> reported a case of acromegaly in which a sarcoma of the pituitary body was found. Some cases of acromegaly have been reported where no apparent disease of the pituitary body existed. Virchow's<sup>3</sup> case belongs to this group, while, on the other hand, many cases of pathologic pituitary gland have been reported in which no signs of acromegaly were demonstrable. Thus F. A. Packard<sup>4</sup> reports a case where the pituitary gland was enlarged and no signs of acromegaly were present. Lloyd<sup>5</sup> reports a case of tumor of hypophysis showing no phenomenon of acromegaly. Dercum<sup>6</sup> cites a case of Weir-Mitchell's in which an aneurism of an anomalous artery occupied the cavity of the sella turcica and had destroyed the pituitary body without producing symptoms of acromegaly.

Packard, Lloyd and Dercum question the rôle of the pituitary gland as a factor in the development of acromegaly. With few exceptions, however, most cases of acromegaly reported have shown

disease of the pituitary body in one form or another; it is, therefore, fair to assume that the pituitary gland is the main disturbing element in the development of acromegaly. Whether disease of the pituitary body *per se* is the cause of this condition or because of disease of this gland other ductless glands are so influenced that their secretions are altered to the extent of causing this peculiar hyperplasia, I am not prepared to say.

**CASE REPORT.**—Michael Evanoff, white; age 34; nativity, Bulgaria; occupation blacksmith.

*Previous History.*—Knows nothing of his antecedents except that his parents were strong and healthy. Patient was in perfect health until one year ago, at that time he experienced headaches every evening when he returned home from work. Six months ago he began to have pain in his back in the region of the tenth dorsal vertebra, this pain travelled upward to the nape of the neck; continued at his work in spite of the pain until one week prior to his admission to the Philadelphia General Hospital.

*History Since Admission.*—Admitted to the tuberculosis department of the Philadelphia General Hospital on November 24, 1917, complaining of slight cough, very little expectoration, occasional night sweats and pain in lumbo-dorsal region.

Physical examination, on admission, made by interne:

White male of small stature, though of good muscular development. Head, eyes, ears, nose and mouth negative. Neck somewhat short. Chest: Slight spinal curvature. Lungs: Infiltration of both apices. Heart: Apex beat displaced towards the left. Abdomen: Liver somewhat enlarged. Stomach, spleen and kidneys in their normal positions. Extremities: Both wrist and ankle joints enlarged, otherwise negative.

Physical signs taken by myself on March 20, 1918, about three months after admission, showed the following:

White adult male, height 5 ft. 4 in. Weight 120 lbs., lost ten pounds since admission. Skin somewhat coarse, no discoloration, mucous membrane negative, no oedema, musculature flabby. Distended veins in both forearms, no other venous distention. Head round, rather small in comparison to face. Ears small, no signs of otitis media.

Eyes: Orbita deep, prominent supra-orbital ridges, slight promi-

nence of malar bones, conjunctivæ pale, pupils equal and react normally to light and accommodation.

Nose long and very prominent (increase of cartilagenous structure), measuring from root to tip 8.5 cm. Across alæ 8 cm. and across the bridge 7 cm. Mouth: Lower jaw prominent, lower lip full and protrudes beyond upper. Gums in good condition, slight separation of teeth in upper and lower dentures. Neck very short, caused by rising of shoulders. Thyroid slightly palpable. Increased dulness over upper part of sternum would indicate persistent thymus. Chest is slightly emphysematous, epigastric angle is obtuse. Posteriorly, distinct kyphosis corresponding to second to eighth dorsal vertebræ. Curve is 17 cm. A plumb line dropped from the apex of the curvature extends 2.5 cm. from the body. Circumference of chest at greatest point 87.5 cm. Circumference of chest on level with both nipples 83 cm. Lungs: Infiltration of both apices.

Heart: Apex beat in the fifth interspace below the nipple line, cardiac dulness increased across the sternum, heart sounds regular, rapid and of good volume, slight accentuation of second pulmonic. Abdomen is somewhat distended, no tenderness or rigidity, liver and spleen not enlarged, kidneys not palpable.

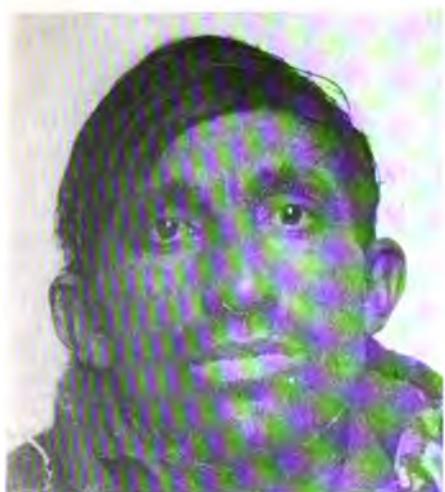
Upper extremities: Shoulder and elbow joints not enlarged, wrist joints enlarged, hands broad and spade shaped, fingers short and thick with curved or rounded nails, having a tendency toward clubbing.

Lower extremities: Hip joints normal, knee joints enlarged (inner condyles). Ankles and lower portions of tibia very large, toes are all symmetrically enlarged.

X-RAY REPORT.—Lungs: Tuberculous infiltration of left apex, also involvement at right upper lobe, considerable thickening about hylus. Right side of heart is enlarged.

Bones: Periosteum is thickened along the metatarsal of great toe, also along the first phalanx adjoining the toe and along the shaft of metatarsal of fourth and fifth toes. A similar condition exists in all the phalanges of the hand and to a lesser extent in the metatarsal of the fourth and fifth. There is an absorption of interarticular cartilage between the third and fourth dorsal vertebræ, so that one bone rests directly on the other, with some tilting of the third dorsal. The femur also shows some periostitis of the shaft.

FIG. 1.



Michael E. Philadelphia Hospital. Acromegaly, showing massive features.

FIG. 2.



Michael E. Philadelphia Hospital. Acromegaly, showing massive nose, protrusion of lower jaw, and prominent orbits.

FIG. 3.



Michael E. Philadelphia Hospital. Acromegaly, showing curvature of spine and shortness of neck.

FIG. 4.



Case of emphysematous chest. Philadelphia Hospital. Compare this gradual curve of spine with the acute curve in the case of acromegaly (see Fig. 3).

FIG. 5.



Showing characteristic enlargement of fingers (with round nails), toes and tibia.

FIG. 6.



Case of pulmonary hypertrophic osteoarthropathy, showing characteristic clubbing of fingers, enlargement of ankles and of great toes. (Compare with Fig. 5.)

FIG. 7.



Acromegaly, showing periosteal thickening of phalanges and enlargement of soft parts.

FIG. 8.



Michael E. Philadelphia Hospital. Acromegaly, showing periosteal thickening along metatarsals and increase in soft parts.

FIG. 9.



Michael E. Philadelphia Hospital. Acromegaly, showing posterior clinoid hypertrophied, pushed upward and somewhat eroded. Anterior clinoid enlarged. Small calcified mass apparently in pituitary. Shadow above sella probably that of tumor.

FIG. 10.



Michael E. Philadelphia Hospital. Acromegaly, showing posterior clinoid hypertrophied, pushed upward and somewhat eroded. Anterior clinoid enlarged. Small calcified mass apparently in pituitary. Shadow above sella probably that of tumor.



**Head:** Posterior clyloid is hypertrophied and has been pushed upward and is eroded. The anterior clyloid is somewhat enlarged. The sella is normal in size and the floor is intact. There is a small calcified mass apparently in the pituitary; above the sella there is a distinctly circumscribed shadow which is undoubtedly a tumor. The erosin and upward position of the posterior clyloid is also added evidence that tumor of hypophysis exists. The interesting features of this case are:

1. The clubbing of the finger nails, which is unusual in acromegaly; as a rule the finger nails are small and flat, while this case presents well rounded finger nails which have a tendency towards clubbing. Compare the photograph of the hands of the acromegaly patient with those of the case of pulmonary hypertrophic osteoarthropathy.

2. The acute onset of the enlargements of the fingers and toes. When the patient entered the hospital his hands were of normal size. He wore a ring on his ring finger which he took off and gave to the nurse for safekeeping. She advised him to wear the ring, as that would be the safest place for it. When I saw him two months after his admission, the ring was so imbedded in the flesh of the ring finger of the left hand that it had to be sawed off in order to prevent necrosis of his finger. The patient also states that six weeks after entering the hospital he noticed one morning that his hands had become swollen so that he could not close his fist nor manipulate a knife and fork and that this swelling became a little larger each day and that at the end of a week they were as large as they are now.

3. The size of his fingers and toes vary in circumference at different times, and when his fingers and toes are largest he voids larger quantities of urine than when his distal extremities are smaller.

There is no evidence of oedema or elephantiasis.

4. Calculus in pituitary, as shown by X-ray.

#### BIBLIOGRAPHY.

1. Dean D. Lewis, Johns Hopkins Hospital Bulletin, vol. xvi, No. 170, May, 1915.
2. Transactions of the Pathological Society, London, 1898.
3. Berliner Klinische Wochenschrift, xxvi, page 81, 1889.
4. American Jour. of Medical Sciences, June, 1892, page 657 (3rd case).
5. Philadelphia Pathological Society, May 25, 1899.
6. American Journal of Medical Sciences, March, 1893.

## A CLINICAL AND PATHOGENIC STUDY OF FAMILIAL CHOLEMIA

By PAUL REY, M.D.

Grenoble, France

---

SIMPLE familial cholemia is defined by Gilbert as "a pathologic state characterized by a more or less pronounced yellowish tint of the integument, either general or predominating in certain cutaneous areas, without elimination of biliary pigments by the urine, but with their constant presence in the blood-serum."

In this definition the expression *pathologic state* is employed but to me it seems much exaggerated. Cholemia *per se*, is not an extraordinary phenomenon. There is a normal physiological cholemia just as there is a pathological cholemia from a highly exaggerated state of the physiological status in certain pathologic conditions of the liver. Simple familial cholemia is an intermediary state between the two extremes; it is, one might say, a special physiological manner of being of the hepatic gland, or in other words, it is much more a temperament than a true pathologic process.

Cholemia is an hereditary familial affection and by questioning these subjects it is easy to obtain the history of various symptoms either in the ascendants or collaterals. Its familial character has such importance that Gilbert and his school have designated this special state as *simple familial cholemia*. When one has the good fortune to trace the trouble back to its origin, it will usually be found that this temperament has existed in the subject since birth. In some cases overwork or infections, severe mental emotions, have been supposed by the patient to have been the starting-point of his ills, but I am inclined to believe that these are merely purely secondary causal factors, which only were the means of attracting the patient's cholemia to himself or family, the trouble having been overlooked until then. Therefore they are negligible. But a careful interrogatory will show that before the effect of these secondary causes, the cholemia had made its existence evident by disturbances which were not attributed to it.

The patient will present a yellowish tinge of the skin, sometimes very slight, with a trifling periocular dark shading. There may be numerous pigment spots on the forehead and here and there on the face are some nævi. The patient will complain of various disturbances which will be considered further on in detail, which establish the fact that he was cholemic a long time before his attention was drawn to his condition by the appearance of an icterus. It is heredity which presides over the appearance of cholemia and if the patient's antecedents are carefully gone into, one will always discover in the ascendants some one of the various manifestations of the biliary family, such as angiocholitis or angiocholocystitis, of the catarrhal or occluding type.

In the opinion of Gilbert this heredity is a crossed one and on account of the large number of these cases which have come under his observation his opinion is of unquestioned value. This is not surprising, because heredity often has inexplicable caprices. Thus, in hæmophilia the male subjects only transmit the diathesis to their descendants of the female sex and they in turn do not realize the hæmophilia but only pass it on to their male offspring.

As far as sex is concerned, cholemia seems to have no preference, both males and females being equally affected, but as to the race, Gilbert has observed that Jews have a remarkable aptitude for developing familial cholemia. For many years observers have known that climate has a repercussion on the number of diseases of the liver, which are much more frequent in warm climates.

Going still further into the etiology of familial cholemia, one perceives that there is an almost unique factor which dominates it, namely heredity. This factor should be known in its efficient causes. This functional taint of the hepatic cell I regard as a determining factor of cholemia. From its manifold functions, from its relations and connections, the liver is obliged to submit to very numerous pathogenic causes. Thus affections of the stomach, intestine, spleen, pancreas, peritoneum and even the pleura occupy important places in hepatic pathology.

The liver is particularly sensitive to certain toxins and poisons, such as the tubercle bacillus and alcohol and above all fermented drinks exert a nefarious influence upon it. In diseases of the nutri-

tion, so numerous and heterogeneous, the dominating etiological factor—a defective diet—is not without having a remarkable nocuity on the liver. One need only recall the unhappy effects of a diet too rich in albuminoid substances and Murchinson long since insisted on the fact that fatty and sweet substances, when consumed in only small quantity, have as a result, a disturbance of the hepatic functions. An exaggerated alimentation, the repeated use of spicy foods, containing the essential oils, rarely omit the production of liver troubles, while the abuse of falsified foods and preserved edibles possess a similar action.

Finally, in this most complex heredity, should not a place be given to the exigencies of modern life, which, from the continually increasing difficult problems to which it gives rise, is not without creating a state of cerebral tension, a kind of fever of intellectual overwork? —and it would be useless to deny the unhappy repercussion on the antitoxic functions of the hepatic glands resulting therefrom. All physiologists admit the peculiar nefarious action of cerebral toxins.

These considerations lead to the recognition in familial cholemia, that there are other more general causal factors derived from the domain of sociology beside the causes belonging to pathologic heredity. The former may, perhaps, explain the greater frequency of diseases of the liver, which seem at present to be decidedly on the increase, at least in France.

In the fact of heredity it should not be forgotten that cholemia is a familial affection, that is to say that in the patient's forbears there will be found numerous examples of biliary disturbances, but its pathogenesis is not as yet clearly understood. That an important factor in this morbid process resides in the bile ducts, no one denies, and the positive pathologic findings of Gilbert show the logic of this hypothesis.

Under the influence of various causes, and they are many, susceptible of changing the hepatic functions, the normal cell is soon disturbed in its equilibrium and physiology, either temporarily or permanently. The cholemic is a patient who carries the burden of a heavy heredity and since his hepatic cells are not normal, the cholepoïetic function is disturbed. It may be that the liver elaborates a special bile, possessing physico-chemical properties different from that of physiologic bile, an hypothesis which experimental physiology, re-

gardless of its immense progress, cannot verify even in the present enlightened state of our knowledge. There clearly must be some process besides angiocholitis to account for familial cholemia.

If Gilbert's hypothesis is strictly adhered to, a permanent infection of the biliary tract must be supposed to exist and even if this be admitted, this infection must have a mild evolution. Unquestionably, a typhoid fever, a defective diet or the development of a still much-discussed protozoair in the biliary tract, must modify the cholemia and produce a temporary or permanent icterus, but then the process becomes a catarrhal icterus, a lithiasis or a neoplasm in a cholemic subject.

Now, a cholemic person usually supports his affection well, so that some complication becomes necessary in order to darken the prognosis of the diathesis. An angiocholitis results in icterus from some obstacle preventing the normal flow of bile, so that it is clear that the liver of a cholemic must hypertrophy, just as the right heart does in the case of a long-standing bronchitis. But this does not occur and alone an angiocholitis would tend to make a cholemic become an acholemic with jaundice, a retention icterus or an orthopigmentary icterus.

Researches have been carried out which show that in many cholemics there is urobilinuria, so that these data place a simple acholemic icterus in the class of icterus from secretory disturbances or metapigmentary icterus. Therefore, it is hardly possible to refuse an important part in the formation of cholemia to the hepatic cells.

This conception may make certain symptoms more comprehensible than the angiocholitis by itself can explain satisfactorily. If one compares cholemia to renal sclerosis, for example, two stages may be assigned to it, the first being a long one of compensation, during which the pathologic state is well borne by the patient. The second is that of rupture of the equilibrium and the resulting complications. During the period of compensation, when cholemia often necessitates a blood examination in order to diagnosticate it, so poor and sometimes so fleeting is its symptomatology, hemorrhages are often observed. Usually the loss of blood takes place in the form of epistaxis (of growth so-called) or a menorrhagia erroneously termed essential,

terms which only serve to conceal our ignorance of the pathogenesis of this diathesis. Gilbert admits the hepatic origin of the hemorrhages and considers them as an important symptom to be looked for, and he even goes so far as to perceive a close relationship between cholemia and haemophilia.

Undoubtedly, hemorrhage from the nose or gums, haematemesis, malæna, purpura, etc., are each manifestations which form part of the usual clinical picture of diseases of the liver, but they only occur as distant complications at a time when the normal physiology of the hepatic parenchyma has become thoroughly disturbed. This is so true that unquestionable results obtained from pathological researches have shown that these various forms of hemorrhage, although met with at the beginning of a cirrhosis, in reality correspond to a cell lesion histologically appreciable, but whose restricted extent prevents a clinical diagnosis from being made.

Another proof of the functional trouble of the hepatic cell is furnished by the examination of the urine. In choleemics, the existence of disturbances of the hepatic chemism is revealed by digestive glycosuria and hypoazoturia. Finally, the violent megrim frequently met with in choleemics may, perhaps, be accounted for by a functional disturbance of the liver, which plays such an important part in the neutralization of toxins.

The absence of hepatic hypertrophy, the presence of urobilinuria, early hemorrhagic symptoms and various disturbances of the hepatic chemism, all seem to show that a mild ascending infectious angiocholitis is not the only process in cholemia and that in the pathogenesis of this morbid process a large share should be accorded to functional disturbances of the hepatic cell. The hypothesis of an angiocholitis cannot be rejected, since we have the proof of its existence, but it may be assumed that the functional disturbance of the hepatic parenchyma is otherwise important and plays a decisive part in the pathogenesis of familial cholemia.

If this pathogenic conception be not admitted, the type described by Gilbert would only be a sub-class of cholemic hepatism, a cholemia from an angiocholitis. In other words, in the cholemia class, a distinction would have to be made between a cholemia from angiocholitis, biliary lithiasis, from functional disturbances of the liver, excessive

haemolysis, etc. Such a classification might be theoretically satisfactory, but could hardly be so from the clinical viewpoint.

We know the effects of this functional disturbance of the hepatic cell and it would be of the greatest interest to understand its mechanism. Is there hypohepaty or hyperhepaty? The latter physiological status would seem more probable, but it still remains to be demonstrated. An excessive haemolysis which some writers maintain exists should not be ignored and merits, with other hypotheses, to be examined.

To my pathogenic conception it may be objected that it is wanting in proof, while that of an angiocholitis is based on lesions observed. The pathological data acquired by Gilbert in no way injures my hypothesis. In point of fact, what physiologist today can detect a minute functional trouble of the hepatic cell and give precise rules for diagnosing it surely?

In the *Dictionnaire de Médecine*, edited by Adelon (edition 1844), the following lines are to be found: "The bilious temperament has as a principal character, not the quantity of biliary secretion, although the action of the liver is here greater than in other temperaments, but the dark tint of the pigments of the skin, eyes and hair. People of this temperament have black, stiff hair, dark or black eyes, a yellowish-brown skin, a very developed and stiff pilous system, a physiognomy offering the marks of a pronounced character and expressing firmness and intelligence. The frame is strong without unnecessary fat, the muscles are vigorous although not large. The principal viscera, are quite well developed," etc.

Individuals of this temperament generally are intelligent and have much capacity, the sensations and passions are intense and durable. They have great ambitions and are obstinate in satisfying them. These are the characteristics of great men.

What predominates in a cholemic subject is a rather yellow tint of the skin, sometimes imperceptible, and a particularly intense pigmentation. The pigment spots are scattered over the forehead (chloasma hepatica), while here and there are a few naevi. Of course there are subjects who in no way recall the type of acholemic. Some of these subjects are apathetic, neurasthenic, hypochondriac or very nervously irritable. But with all these different states of the nervous

system, there is a common basis in these patients and that is that they present identical symptoms which I shall now consider.

In order to properly develop this analysis it is necessary to examine successively the various changes wrought by cholemia on each system of the body in particular. This is the only means by which one can distinguish a particularly weighty symptomatology. We will first consider the *fundamental symptoms* and after these the *secondary symptoms*.

*The fundamental symptoms concern the state of the integuments, the urine, the serum and the liver and spleen.*

In cholemia a vulgar icterus with impregnation of the conjunctivæ is never to be looked for as it will rarely be found. There are other signs which will draw the physician's attention, such as xanthodermies, melanodermies, xanthelasma and finally arterial or capillary nævi. Xanthodermia in these subjects is not that of ordinary jaundice; it is a concealed icterus with absence of yellow conjunctivæ. Generally the face is more involved than the remainder of the body and it is common to compare these subjects to creoles or Oriental people. This icterus may be localized to certain points of election such as the palms of the hands and feet, the so-called palmo-plantar icterus.

But the skin may transform the biliary pigments instead of eliminating them by exfoliation and retains this melanin in its layers and then a melanodermia results. According to the case the process is uniformly distributed or else localized. Among the latter are pigmentary nævi (beauty spots), freckles or biliary spots; the periocular dark shade and a distinct pigmentation of the forehead contributes to make a real biliary mask in every way comparable to the chloasma of pregnancy with which it is often associated. Xanthelasma of the eyelids is also useless to note but a tiny xanthelasma occupies the angle of the lid which is easily overlooked. To these cutaneous signs are to be added the frequent presence of arterial or capillary nævi, which have a semeiologic value.

*The Urine.*—Usually there is a pigmentary acholuria but it must be pointed out that there is also an intermittent choluria. The cholemic pigments are not all transformed into melanin, the cutaneous syndrome of cholemia does not entirely absorb them. Gilbert has shown that in the great majority of cases urobilinuria exists and better

still, he has demonstrated that there is a close parallelism between the cholemia and the urobilinuria, whose respective proportions in the blood and urine varies in close relationship.

*The Blood.*—The serum contains pigments and is the diagnostic point of the affection. The red-blood corpuscles and leucocytes in the first place increase, reaching a very high maximum and then slowly diminishing. The red-blood cells diminish both in number and globular value, which sign is a serious argument for those who maintain that the starting point of familial cholemia is an excessive haemolysis.

*The Liver and Spleen.*—Usually there is no appreciable change to be detected by examination, but a hepatomegalic and splenomegalic form of the affection has been described although the process is devoid of any painful manifestations on the part of the viscera involved.

The various hemorrhagic manifestations which are directly related to cholemia must not be overlooked among the fundamental symptoms of the process. Epistaxis, haematemesis, melena and haematuria are often met with in cholemic subjects. Purpura is prone to accompany hepatic diseases and this frequency of, and tendency to hemorrhages in hepatic cases is a well known fact.

These hemorrhages are such an important symptom that in certain cases Gilbert confounds haemophilia with cholemia.

The secondary symptoms are those which often attract the most attention and which give to each cholemic subject a particular aspect and they also serve to establish different types of cholemia. I think that we may distinguish five principal types: *the common form, the cutaneous form, the gastro-intestinal form, the nervous form and the hemorrhagic form.*

Certain facts tend to show that a rheumatismal and a chlorotic form may exist, but in my opinion there must be two pathologic states, two distinct diatheses in these special cases.

Nothing need be said of the common form of cholemia as its name is its definition. The cutaneous form is recognized by a more intense xanthelasma, a melanodermia similar to Addison's disease, a tenacious pruritus, a recurring purpura or prurigo.

The gastro-intestinal form includes the numerous cases of hyper-

petic dyspepsia (pseudo-gastric ulcer) and enteritis and to this a hemorrhoidal form might be added.

The nervous form comprises all those states in which somnolence, melancholia, hysterical and neurasthenic tendencies or hypochondria are noted.

Finally, in some cases cholemia assumes a distinct hemorrhagic aspect and the diagnosis may be particularly difficult between it and that of haemophilia. But in reality one must not be led to conclude these special forms, so different in their manifestations, are met with with such distinctness. Usually, the symptoms of one form will be mingled with those of another, so that associated secondary phenomena will be so important that they in themselves will make the diagnosis evident.

*The Pathologic Physiology.*—The question as to how the functions of the liver are carried out in simple familial cholemia is all important. For Gilbert and his school nothing is easier to explain, namely, that there is a constant presence of bile pigments in the blood-serum, without elimination of these pigments in the urine. In a word, there is cholemia without choluria.

In his remarkable work: "*Traité de physiopathologie clinique*," Grasset treats this question as follows: "The bile passes directly into the blood by the vessels and especially immediately by the lymphatics giving rise to cholemia and its consequences.

"The hemolytic action of the bile has been known since Hunefeld (1840). Cholemia causes a special anemia; a diminution in the number of red corpuscles and an absolute elimination of the hemoglobin with a manifest increase in the resistance of the red corpuscles to the dissolving action of distilled water and, in a lesser degree, to the dissolving action of sodium taurocholat; persistency and then manufacture of more resisting globules from a kind of cell and humoral immunisation against the action of distilled water.

"In other words, when the bile reaches the normal blood, the latter assumes the defensive. We shall see further on that it is not the same when the initial lesion is in the blood, when hemolysis precedes and produces the icterus (hemolytic icterus).

"The quantity of leucocytes is occasionally slightly increased, but usually normal or decreased.

"This nefarious action of the bile on the blood has been attributed to pigments (Bouchard and de Bruin) while at present it is attributed rather to the bile salts (Dresch).

"This biliary intoxication sets up multiple reactions in the hematopoietic system; the spleen particularly, increases in size, sometimes very much so, with exaltation of its hematolytic and pigmentopoxic function; the hematophagia is the consequence of the biliary impregnation and of the consecutive hemolysis.

"The repartition and final end of the pigments in cholemia have been studied; some are fixed, others are eliminated by various external secretions, particularly the kidney (although the larger part of bile injected into the blood or connective tissue returns to the liver), from which results renal lesions well studied by Goujet and due above all to the bile salts."

Therefore, at present cholemia is not discussed, because the changes it causes in the composition and manner of being of the blood serum are recognized by all. But where the difficulty begins and the problem becomes complex, is when the future of the cholemic pigments is discussed, because these pigments cannot remain indefinitely in the serum. It happens, of necessity, at a time when they are eliminated by the emunctories unless they be, after or without transformation, deposited in some part of the organism (?) like the ochre pigment becomes deposited in the meshes of the splenic tissue. The blood admits only temporarily various bodies, such as  $\text{CO}_2$ , toxins, urea, uric acid, sugar, etc., and this liquid tissue rejects those impurities at a given time. Were this not so for the cholemic pigments there would not be cholemia but icterus, a fatally progressive and slowly evolving icterus. Now, icterus gravis of the progressive type is met with in cholemia only as a rare complication, or at least a late one.

For Chauffard, acholuria is possible. "Cholemic subjects are cholurics or at all events, hypochlorics." Such is his conclusion and in order to reply to these objections, Gilbert undertook a series of researches which showed that in the majority of cholemic subjects urobilinuria was present. Gilbert, it is known, maintains that urobilin does not exist in a preformed state in the blood, but is the result of the transformation undergone by the normal bile pigments in the

renal filter. It is on account of its hydrating and reductive powers that the renal gland transforms bilirubin into very diffusible urobilin.

Gilbert further remarks that "it was necessary to ascertain, if in familial cholemia, a rather marked amount of urobilin would not be found, resulting from the transformation in the kidneys of the bile pigments contained in the blood serum. From the fact of this transformation, there would consequently be acholuria and urobilinuria."

"I have been able to show the presence of urobilinuria in almost every case, and urobilinuria and cholemia have a similar relationship; there is parallelism. Therefore, cholemic subjects are acholuric but urobilinuric."

Gilbert goes still further, since in the same paper he declares that the search for urobilinuria will allow one to make a diagnosis of familial cholemia before examination of the blood-serum is made. As to the various pigmentations which are rarely wanting in the symptomatology of cholemia, they result from the transformation of bile pigments into melanin in the epidermis.

Gilbert's conclusions, which are the outcome of very detailed and thorough researches cannot be held up to doubt. Nevertheless, it seems to me regrettable that, even in his most recent writings, he limits himself, when he defines cholemia, to opposing cholemia to acholuria, without saying that this acholuria is replaced by urobilinuria.

*Prognosis and Diagnosis.*—The prognosis of cholemia depends almost entirely on the patient himself. If he is sober, temperate and will submit to a diet which is often severe it can be said that the outlook *quoad vitam* is assuring and that the patient will in all probability remain in a state of compensation and free from any serious complication.

If, on the contrary, the patient is a high liver and consumes defective food and if he will indulge in alcoholic excesses, even if only intermittently, if in a word he will not submit to the strict rules required by his temperament, the prognosis is not good since numerous and varied complications are bound to occur, from temporary catarrhal icterus, which is usually benign, to icterus gravis or neoplasia, neither one of which ever pardons.

It is almost unnecessary to point out that in practice it would be interesting to be able to make a diagnosis of so important a state as

this in order to avoid those complications to which it inevitably leads if the diathesis is not properly treated. When present in sufficient number, the various symptoms which give a clue to the real condition will make the diagnosis positive. But as I have said, cholemia is rather more of a temperament than a disease and frequently these patients suffer no inconvenience or at any rate the disturbances of which they complain are so indistinct and so difficult to classify, that they may not be attributed to their real cause. Therefore, a sure pathognomonic sign must be sought for which will be the touchstone of the condition and this sign is the cholemia.

Alone, examination of the blood-serum will clear up the diagnosis in any case of doubt. In the normal state there is a cholemia and the blood-serum contains an average quantity of 1/36500 of urobilin, or 2 centigrams to each 7 liters of serum and 8 centigrams for total mass of blood in the average person.

In a summary of his analyses of the serum of 60 cholemic patients, Gilbert came to the conclusion that in familial cholemia this proportion is more than twice this amount. It is 1/17,000, or 5.9 centigrams per liter of serum and 18 centigrams for the total mass of blood.

Here, therefore, is an exact means by which familial cholemia can surely be recognized and it is this means that will settle the question in doubtful cases. It is an approximate means but nevertheless sufficiently accurate, since, according to Gilbert, the blood mass contains 45 centigrams of bilirubin in chronic icterus and 1 gram in biliary chirooses.

It may not be out of place to recall at this point the conclusions of Stankewitch's thesis (Paris, 1904), on a procedure of dosing bilirubin in the blood-serum by means of cholemimetry of Gilbert, Herscher and Posternak. They are as follows:

(1) Guelin's reaction, characteristic of the presence of bilirubin, consists, when one operates on a non-albuminous fluid, in an ensemble or succession of colored rings first described by Guelin and Frerichs.

(2) This same reaction, made in an albuminous fluid, is quite different and offers the following peculiarities: (a) the series of color rings of Guelin appears only in fluids rich in albumin under the influence of nitric acid if the content in bilirubin reaches or extends beyond a certain degree of concentration, namely, 1/3500 in a thick-

ness of 1 centimeter; (b) below this figure, the aspect of Guelin's reaction will vary according to the concentration of the bilirubin: with 1/7000 there will be a blue ring with a greenish reflection (Hayem's reaction); below 1/11,000 a blue ring with a violet reflection (Gilbert, Herscher, Posternak); (c) a concentration of 1/40,000 corresponds to the limit of Guelin's test which remains negative below this figure.

(3) The blue rings with greenish reflection of Hayem and the blue with the violet reflection of Gilbert, Herscher, and Posternak, produced by nitric acid in albuminous media, are quite as characteristic of the presence of bilirubin as the series of Guelin's color rings.

(4) These same blue rings observed in certain blood-sera should be attributed to the presence of bilirubin and in no way due to other coloring matters.

(5) The limit of reaction of Guelin permits making the estimate of the bilirubin content of the blood-serum and forms a basis for the procedure of Gilbert's cholemimetry.

(6) In this procedure the serum to be examined is diluted with varying amounts of artificial serum so as to obtain a mixture giving exactly the reaction of Guelin's limit. To obtain these mixtures the dilution should be made by the volumetric method. Knowing the quantity of the initial serum contained in the mixture at which the limit reaction is observed, the content in bilirubin of the serum to be examined is calculated according to the formula:

$$x = \frac{10 + a}{a} z$$

in which  $x$  is the concentration desired,  $a$  = the quantity of initial serum added to the mixture and  $z$  the constant estimated at 1/40,000.

(7) Gilbert's cholemimetry is easily of clinical application, being so simple, and has given very important data.

*Treatment.*—This must be pathogenic and should fulfill two indications, namely, to act against the slight ascending infectious angiocholitis and secondly, to attenuate the functional disturbances of the hepatic cell as much as possible.

To treat the angiocholitis is at present an easy matter for there are numerous drugs which have the property of being eliminated by the biliary tract and which possess well-known action on the bacterial

flora, both normal and pathologic. To this end the various compounds of benzoic and salicylic acids are to be tried in turn. Sodium benzoat and sodium salicylat given in combination according to Chauffard's formula, give remarkable results.\* Lithin salicylate may, in some cases, be of value.

Finally, urotropin is one of the best antiseptics of the biliary tract as has been shown during the past decade by reports from many countries. Calomel also is an antiseptic of unquestionable value on the hepatic gland.

But if, as I admit, simple familial cholemia is related to some functional disturbances of the liver; if this functional derangement of the hepatic gland is due to a hyperhepaty, which is the most probable hypothesis, it is certainly evident that the principal point in treatment of cholemia is to address the treatment to the functional derangement with the object of reducing it to its narrowest limit by relieving the physiological functions of the gland as far as possible. It must be admitted that, with the exception of various cholagogues which are to be resorted to at a timely moment, our therapeutic arsenal is very poorly furnished and there is not a single mendicament which has the power to modify the hepatic functions. However, the functional disturbances may be lessened in their manifestations.

The hydromineral treatment is always temporary. To combat the cholemia, which is a chronic pathologic state, a daily and persistent action is essential, and diet, only diet, can fulfill this indication. However, a hydromineral treatment should always be essayed and usually it will have excellent results on the cholemia by attenuating its effects.

---

\* Note.—Chauffard's formula is:

B.

Sodii benzoat..... 20 grams.  
Sodii salicylat..... 10 grams.

---

M. Div. in cachets No. XXX

Dose.—Two to three cachets a day, to be taken between meals with a wine-glass of Vichy-Celestins.

This is to be found in Chauffard's recent book: *Le Cours sur la Lithiasis biliaire*, Paris, 1914, a book replete with excellent teachings, interesting from beginning to end, and well worth reading. [Translator's note.]

Cures at Evian, Vittel, Contrexeville, Pouges, and particularly Vichy, will be of great utility.

The liver is an antitoxic organ, therefore it is important to eliminate all toxic matter from the food, at least as far as this is possible. The liver forms glycogen and regulates its distribution, therefore the materials to be transformed must be reduced. The liver fears nitrogenous substances when in excessive quantity, it is particularly sensible to certain poisons, among which are alcohol and especially fermented drinks, therefore these should not be allowed.

By eliminating so many things there hardly remains but a lacto-vegetarian diet and, in fact, this is the only one that should be permitted. A cholemic subject should abstain from alcohol in every form, likewise meat in any great quantity, spices, preserved goods and salted foods, sausages and the like.

In beginning this alimentary hygiene the patient should be first put upon a cure of skimmed milk for about three weeks on the condition that a rest cure is combined with it, because such a reduced diet is incompatible with any active physical exercise. Kafir may be substituted should milk not agree.

In the second phase of the regimen the diet may be increased to a few little meals a day but the skimmed milk should form the principal article of diet. At noon and at night some form of milk soup, underdone eggs and stewed fruit may be allowed.

Then the diet is again increased, although retaining its lacto-vegetarian character. It must be moderate in quantity because the liver would be overcome again by a too intense alimentation and a diet too rich in albuminoids and carbohydrates would be badly tolerated. Fats particularly are badly assimilated so that they should be only allowed in moderation, even in the preparation of food. Eggs must not be indulged in too freely, particularly the yolk.

Well cooked white meats are to be preferred as they are easier of digestion. Other than the white meats, fresh white fish and vegetables can be allowed but those that are difficult to digest should, of course, be abstained from, such as cabbage, tomatoes, mushrooms, etc., and this also applies to spices, acid and raw foods.

Massage of the liver has been recommended at the time when opsiuria, the one early symptom of portal hypertension, is first ob-

served. These are the cases which offer a slight increase in the size of the liver and when this is discovered an analysis of the urine should at once be made.

Opotherapy has become very popular and Gilbert has seen much good come from it. The use of hepatic extract acts favorably on the hepatic insufficiency and particularly on the hemorrhages. Biliary opotherapy is efficacious in controlling the disturbances of the intestinal biliary function and the action of pancreatic extract is manifest when there are secondary digestive derangements. The thyroid extract may be beneficial in controlling the pruritus.

## SENILE EPILEPSY, WITH THE REPORT OF FOUR CASES

By O. MATORINE, M.D.

Moscow, Russia

---

THE epileptic syndrome is distinct in its symptomatology and it can even be said that it is only characteristic from its symptomatology. I insist on this fact as it is not my intention to give in detail all the causal factors which may create this syndrome in elderly subjects. Now, since I employ the term *senile epilepsies*, one might infer that there is a difference between this affection in old people from that met with in adolescents and adults and as we use epilepsy in the plural, it might be concluded that there are several different types. Let me explain.

If by epilepsy we understand the epileptic syndrome, we really have no need to make this distinction, because the convulsive syndrome which individualizes epilepsy hardly ever varies be it in the adult, adolescent or the old. It is always a succession of well-known phenomena, aura, tonic convulsions, then clonic, then coma and the period of obnubilation of more or less long duration. Neither does it vary much according to the causal factor present.

On the other hand, should one include in epilepsy all the abnormal phenomena which have been noted in subjects afflicted by this mental trouble from childhood? In this case the limitation of the characteristics of senile epilepsy would be hard to outline, because should the character of senile epilepsy, its metabolism, its reactional reflex, both organic and of relation, diverge from the normal, it would become a difficult matter to decide where epilepsy stops and senility begins. Thus there are senile epileptics who present delirium just like that met with in elderly subjects who are not epileptic, while on the other hand there are subjects who do not become delirious and present no other morbid phenomenon than attacks of epilepsy.

It would seem easy to settle the question by saying that senile epilepsy is an epilepsy characterized by the classic convulsive syndrome occurring in subjects over fifty years of age. But, besides the

arbitrary nature of such a definition, one could in way of contradiction offer the following argument by saying that there are cases of precocious senility presenting the characters of elderly people where the subjects are only 35 to 40 years of age, while on the other hand, there are people who live many years before they can pass legitimately for elderly subjects.

In the description of senile epilepsy which is to follow, I shall at once eliminate those cases in which the attacks did not occur before the age of 45 to 50 years. When one goes carefully into the patient's history one will detect undoubted manifestation of a concealed epilepsy. Some causative factor, such as an infection, a traumatic shock, or acute or chronic intoxication has resulted in the production of an attack of the "grand mal" in a subject who presented all the requisite characters for an epilepsy, excepting just the typical manifestation. Consequently, senile epilepsy is represented by those cases in which the subject reacts to a deviation from the normal state according to the manner of epileptics.

I purposely use the vague expression of "deviation from the normal state," for several reasons of unequal value. We thus imply all there is indefinite and unknown in the multiple links between cause and effect which unite the phenomena of epilepsy and this particular syndrome. I shall endeavor to put in relief what data have been employed for classifying epilepsies into senile and other types, and by this I mean that the patient has conducted himself epileptically, if I may be allowed this term. And lastly, this brings us directly to the classification or perhaps it were better said, to the etiological enumeration of senile epilepsies.

*The Causes of Epilepsy in Elderly Subjects.*—These may be classified as follows: In the first place *cerebral ischemia*, which should be differentiated from arteriosclerosis at least from the fact that arteriosclerosis is a constitutional anatomic condition, while ischemia, often derived from the former process, may appear on its own account without any relation to the atheromatous process.

*Cerebral arteriosclerosis* is so very common that it may be considered, if not normal, at least almost certain to occur in elderly subjects in whom it develops at a more or less advanced age. From the sclerotic arterial changes, changes in the blood-pressure make them-

selves felt more strongly than in individuals possessing young and elastic arteries, resulting in anæmia and hyperæmia more frequently and especially in a more offensive way for the patient who is the possessor, without mentioning miliary aneurysms and the always possible rupture of a small arterial twig in the brain, the symptoms to which it gives rise easily assuming the picture of an epileptiform attack.

In the same order of ideas is *thickening of the cerebral envelopes*, which rather periodically irritate the cortical neuron. These lesions are pachymeningitis and leptomeningitis whose starting point may not of necessity be a senile sclerosis.

Traumatisms of the brain, by the production of chronic compression from cicatrices, old exudates and osseous or connective tissue, are to be included in this etiological group.

*Hemorrhagic raptus* merits a mention by itself. It may generate the epileptic syndrome at the time the hemorrhage is taking place, from irritation at the time or after the blood-clot has formed. It frequently happens the paralysis resulting from the hemorrhage is very distinct at first but is only transitory, and since it disappears very rapidly, it may very naturally be asked if a real hemorrhage has occurred since the anatomical proof is wanting on account of the wildness of the attack. Thus, as Vires has pointed out, the epileptic syndrome is almost completely found in cerebral hemorrhage and in cases where no arterial rupture has taken place, pachymeningitic thickening simply senile in nature being in play.

The various *cardiopathies* are common in elderly people, but they are not of necessity senile in nature. On the other hand, the epileptiform aspect they may assume is not special to this time of life and often they only act by the intermediary of other causal factors which, if not easy of demonstration, may at least be suspected, such as uræmia, etc., and these causes are comprised in another etiological group.

*Pure senility* is a causal factor of epilepsy from the generalized sclerosis that it causes. It is this sclerosis which, reaching the organs of the internal and external secretions, is the indirect cause of changes in the metabolism, of rapid denutrition upon the most trifling perturbing cause of the general equilibrium, and of the epileptic reaction for which no other starting point can be discovered. This is a most important fact which I shall again refer to in more detail. The

epilepsy from pure senility and polysclerosis, becomes an epilepsy from intoxication in every way similar to those types which have of late been studied under this head.

These *intoxications* may be, so to speak, grossly and directly exogenous in origin; I refer to intoxication by mineral and organic poisons, such as lead, mercury or alcohol. It may also be an intoxication from bacterial poisons, such as syphilis. Or the intoxication may be more obscure, such for example as an autointoxication by a multiple and undetermined poison, such as is found at the basis of diasthesic diseases, gout and rheumatism. And it is thus that the question of the internal secretions and toxic epilepsy are united on the common territory of senility.

Before discussing the theory of epilepsy and senility I will report the four following cases, the first one of which was observed in the service of Prof. Vires, the remaining three having been studied in the service of Prof. Mairet. The precise details of Case I, and the exact clinical picture that it offers, have led me to only briefly refer to the deductions that it inspired. Then the relatively recent theory that this case history suggests has made me extremely prudent in deciding its pathogenesis because from the scientific viewpoint it seemed advisable to record the facts in the first place and await other similar cases and future experiments.

Case I.—P. A., female, aet. 70 years, entered the old people's service, November 1, 1907.

*Personal antecedents.*—Had been well during early childhood. Married and gave birth to three children, the first two dying between the ages of 18 and 20 years from pulmonary tuberculosis. The third child is alive, 38 years of age, and well.

Menopause at the age of 51 years without any incidents. At the age of 60 years the first convulsive attacks occurred and coincided with physical changes, the patient becoming very fat.

The attacks are those of generalized epilepsy, namely, paleness of the face, the cry, fall and tonic and clonic movements, stertor and coma. During the attack the tongue is bitten and there is relaxation of the sphincters. The attacks have not altered the excellent general physical state, the patient eats much and is increasing in weight.

The first attacks did not alter the intelligence, memory or psychic and physical activity and they only occurred every six months. Then they became more frequent, always offering the same character of a typical and complete epileptic attack.

Three years after the beginning, in 1900, the attacks changed. After the attack the patient remained stupid and for two or three days she was fatigued, in-

ert and incapable of all physical movement and any intellectual effort. She was indifferent to everything occurring about her. Then the attacks became less frequent, occurring every 2 or 3 months. They were replaced by paroxysms of agitation and delirium but the data obtained on this subject were not very precise.

From 1900 to 1907, when she entered the hospital, the situation was about the same. There were convulsive paroxysms every 3 or 4 months with attacks of agitation in the interval, likewise delirium, physical and psychic inertia. Treatment consisted in the exhibition of the iodides and bromides, regulating the diet and frequent purgation.

1907: The patient is a strong and very fat subject, the general health and physical state excellent. She has epileptic attacks every 2 or 3 months either in the day-time or at night. During the attack she becomes pale, emits a loud cry and rolls on the ground. Traces of former falls are found in the form of ecchymosis and contusions over the elbows, knees, face and temporal region. When on the ground there are tonic and clonic convulsions. The tongue is always severely bitten and the vesical and rectal sphincters relaxed. The paroxysms last from twenty-five minutes to one-half hour. The period of stertor and deep coma last for about the same time.

When the paroxysm is over the patient presents a peculiar type of delirium. She does not know where she is and thinks that she is at her own home. There are hallucination of the sight and hearing; she sees her children and converses with them. She goes about her household duties, talking to imaginary persons who are her friends and relatives at her native village.

In other words, she relives a period of her past life. Her actual life is completely forgotten and in her cerebral workings she returns to the age of 30 or 40 years. After each seizure the phenomenon of reviving this period of her existence is not constant, that is to say, it does not always occur and when it does it revives either a far away or a nearer period of her life. Sometimes the attacks succeed in a very violent form, 2 or 3 a week, but the interval between them is never more than two weeks at most. During the interparoxystical periods the delirium with hallucinations exists, evoking the past life of the subject.

Functional and organic examination reveals a perfectly normal state of all the viscera and systems, so that as far as treatment is concerned, a polybromide medication in progressively increasing doses was essayed but at the end of two months no improvement was manifest, even after the elimination of the chlorides from the diet had been tried.

The treatment was then changed to sodium borate at the dose of 4 to 5 grams daily, but this medication had no better effect on the attacks and delirium. The patient became more expansive and tried to escape in order to return to the country. The general nutrition now commenced to suffer and the tongue was thickly coated. Diarrhoea appeared so that the drug had to be discontinued and medication with chloral hydrat, alone or associated with Méglin's pills,<sup>1</sup> was instituted, likewise without any result.

---

<sup>1</sup> The formula of Méglin's pill of the French Codex is:

R.

Ext. hyoscyam.  
Ext. valerian.  
Zinci oxidaa 1.5 gram.  
M. J. pil. No. XXX.

(Translator's note.)

On account of the atrophy of the thyroid gland and manifest obesity, thyroid extract seemed to us indicated and its exhibition was begun in January, 1908. The general condition then commenced to improve; the patient went out, walked, and memory and intelligence returned. The epileptic seizures became less frequent and violent but the delirious manifestations persisted. The thyroid extract was stopped in March.

March, 1908, to January, 1909: The patient had a seizure about every two to three months, but she had frequent desire to escape which necessitated a constant watch.

June, 1909: The opotherapeutic medication was again tried. The raw thyroid was first essayed, after which thyroidin in tablet form was given. The seizures disappeared and the memory improved.

June, 1910: Thyroid medication had been stopped in November, 1909, and the patient had no attack until June, 1910, when she had a very violent one. In her fall she fractured the acromion. Since then she has been better and knows those about her, although she remains quite weak.

*Case II.—Female, act. 64 years. Senile epilepsy and alcoholism.*

*Personal antecedents.*—Alcoholism, 3 to 4 litres of wine daily, without counting several glasses of liquor that the patient will not admit taking.

*Hereditary antecedents* are unknown.

On July 19, 1914, this patient was brought into the service after an epileptic seizure that had taken place a week before. There had never been a former attack. When she entered the hospital she was completely lost as to her surroundings and in her reason. The patient was deaf. She refused all food and drink and was personally dirty in her habits.

There was a certain amount of paresis of the lower limbs, walking was accomplished by short steps and there was a deviation of the face to the left. The hair was untidy and kinky.

Slowly the patient became better and at length took food and lived in common with the other patients. No attack occurred until September, when on the 17th of the month she was stricken with a seizure at 3 P.M. and which lasted for three days, the subject remaining unconscious. Since this date, she has had several attacks closely following each other but in these she did not fall.

*Case III.—Male, 56 years of age, entered hospital December 18, 1911.* On his arrival the patient presented a physiognomy expressing no particular sentiment. The complexion is earthy and the face is lined with deep furrows. His look attracted our attention on account of a kind of hesitation that he showed, but the patient explained this by saying he was deaf.

Upon questioning him it was found necessary to do so in writing and then he willingly replied. There was no evidence of any delirious ideas and there did not seem to be any very marked dementia, because he recalled all the details of his commitment, which he is perfectly well aware is his fourth.

Likewise, he recalls quite well things formerly acquired, particularly the date of his birth. However, it should be remarked that the psychic processes are slow and impressions are often indistinct, which led us to suspect a slight degree of nervous depression.

The patient also states that he has had convulsive seizures since September, 1908, during which he suddenly lost consciousness and of which he remembered nothing, other than that the tongue was painful after each attack as it was bitten

during the convulsions. The seizures were more apt to occur in the night but he has also suddenly fallen in the day. The attacks do not leave the patient depressed because he can return to his work momentarily interrupted.

A somatic examination shows arteriosclerosis to an advanced degree. The tendon reflexes are sharp, especially on the left. Standing on one leg is difficult. The pupillæ react to light and are equal. They do not appear to react to distant accommodation. Sensibility seems normal, but it must be added that the study of the patient is rendered very delicate on account of the deafness.

Consequently, it would appear from the results of the examination that the case was one with a convulsive syndrome presenting the characters of generalized epilepsy but on account of the development late in life at the age of 53 years, it would seem to point to a symptomatic origin.

From the nature of his occupation, that of a truck-driver, the patient was almost obliged to be a drinker. There are all the signs of an advanced arteriosclerosis so that it seems that we should look for the explanation of the symptoms in this pathologic change. On the other hand, a longer period of observation is necessary in order to determine the nature of the psychic disturbances necessitating the patient's commitment.<sup>3</sup>

The treatment in this case was hygienic and a daily potion containing 1 gram. potassium iodid.

As to the delirium, the following data were obtained: The patient complains of his wife, whom he accuses of having him committed simply to be rid of him. As already remarked, the patient is very deaf, and it appears that one day while at work he fell unconscious and when he regained his faculties he was deaf. He also thinks he heard a voice telling him to kill his wife, but this only occurred during one day and has not recurred.

In July, 1912, the patient was still in the same mental condition and the epilepsy also, although it seemed as if he had become somewhat stupid and that dementia would soon develop. The patient's mother, who died in advanced years, was said to have had "attacks" which may have been epileptic in nature.

Case IV.—Male, about 60 years old, sent from another hospital with the diagnosis of acute mania with hallucinations. He had attacked the attendants and threatened to kill them.

When he entered the asylum he first acted like a subject about to begin a senile dementia with delirium of recrimination. He maintained that he was committed without any reason and wished to work in the open, in spite of the fact that he could hardly remain standing.

There were evidences of advanced arteriosclerosis; the pupillæ were equal and contracted, but reacted to light and to distant accommodation. Reflexes normal.

Some time after being admitted, the patient had some epileptic seizures which were carefully observed and were unquestionably epileptic. After the attacks, during which he sometimes bit his tongue and voided urine involuntarily, the patient was stupid, dazed and replied to questions inadequately and with difficulty. This condition lasted from one to three or four days according to the violence of the attack.

<sup>3</sup> Note.—Since the above notes were taken, the patient has had numerous epileptic seizures which leave him in a state of forgetfulness, contrary to what he affirmed. The attacks are mostly nocturnal, sometimes diurnal, but they are not sufficiently serious nor numerous to prevent the patient from working.

Potassium bromide at the dose of 4 to 6 grams daily, according to the condition of the digestive tract, and cerebral state of the patient, was exhibited.

Later on, following a more violent attack than the previous ones, the patient was put to bed, and the next day he was carefully examined and made to walk. Walking was, of course, more difficult than before and also more uncertain. The patient dragged the left leg and took a point of resistance on the right lower limb. Strength was slightly diminished on the left in comparison with the right side (upper limbs). Nothing of any note met with on the head and neck. The patient was somnolent and the mind cloudy, so that the examination was difficult.

At the end of about a week the unilateral symptoms mentioned above had vanished.

When the stupidity following the seizures had disappeared, the patient began to talk in his delirium about being unjustly confined and wished to work outside in the open. He also had very numerous hallucinations of the hearing. He heard his sister, who led a loose life, under the roof above him and on account of the noise she made he was unable to sleep. This idea was very obstinate and had been present from the time the patient was admitted.

The toxic theory of epilepsy is very generally admitted and besides making it possible to include the affection in the class of diseases due to causes which, even if unknown, are at least recognizable, will, therefore, be more easily curable in the future.

I shall not insist on intoxications of distinctly exogenous origin which may create the epileptic syndrome, as the subject is well known to all. The action of alcohol on the cortical motor neuron does not differ in old age from that in the adult, but in the former the poison acts on a tissue which has lost its physiological suppleness. On the other hand, a subject who gives himself up to alcohol in advanced years is generally an habitual drinker, and it is uncommon for one to acquire a taste for alcoholic drinks after the age of fifty. Therefore, in these circumstances one is dealing with chronic alcoholics realizing epileptic raptus.

On the other hand, the nerve cell being already fatigued from other causes incident to life, is more susceptible than formerly to the poison. It returns more slowly to its normal state and this is a fact which must be seriously taken into account in the study of all nervous syndromes in elderly subjects. Thus it is that one observes that this class of patients remains much longer somnolent and bewildered after a seizure than in younger individuals.

Thus it may be said, without entering into a long and detailed discussion of all the reactions offered by senile epileptics, that exogenous intoxications assume a particular character in them. The very

unbalanced vascular equilibrium to which their brain centres are submitted and the easy and rapid denutrition of these anatomic structures, all have a most characteristic action on the progress and evolution of epilepsy in elderly subjects.

It would appear that dementia, which is a radical weakening of the intelligence, is quickly produced in such patients, while in young epileptics having frequent violent seizures the intelligence is preserved in fairly good equilibrium regardless of all, while the senile epileptic is at the mercy of a few rather mild attacks.

As I have already said, the intellectual numbness which follows the seizure is much longer in disappearing and the intimate causes of the attack are much more minute than in the adult epileptic. I shall refer to the question again when speaking of the treatment.

What has been said of alcohol may be repeated for other chronic intoxicants which are recognized as apt to produce the epileptic syndrome; I refer to lead and mercury. These intoxications are far less frequent, especially the latter.

As to bacterial infections, the toxins to which they give rise offer too many characters in common with poisons, so that any lengthy consideration of this subject is unnecessary.

The endogenous intoxications are much more interesting because their pathogenic mechanism is far more obscure. We, here, are in presence of a difficult explanation concerning both the epileptic syndrome and the other symptoms of endogenous intoxications, which has given rise to much imagination on the part of research workers and a taste for theories in vogue.

Far from rejecting an essay at explanation, it appears to me far more in conformity with a scientific mind to endeavor to make an attempt in classifying new facts in the light of those already known.

One of the intoxications of endogenous origin that is the best known and which is accused of producing epileptiform attacks is certainly uræmia and although this process follows upon infectious diseases and intoxications of obscure etiology, such as pregnancy resulting in eclampsia, it is none the less most frequently an affection of old age. This is not the place to treat the subject of uræmia and its causes and I only desire to point out that many disturbances with

epileptic characters and many manifestations following cardiac lesions have uræmia as a near relation.

No matter the type of the seizure, be it great or small, vertigo and "grand mal," uræmia may produce them all. In elderly subjects uræmia will cause epileptic phenomena much more readily than in adults and this is important to remember from the standpoint of treatment.

Another endogenous intoxication is the gastro-intestinal and in this process the liver plays an important part. The viscus no longer neutralizes or transforms with sufficient energy the poisons brought to it from the intestinal or digestive tracts and the convulsive attacks may be the index of the hypofunctioning of the hepatic gland.

There is also an autointoxication which, more than any other, merits its name. It is that which results from the cessation of the function of the functional deviation of the glands having an intense secretion, and the question arises as to what way these factors can act in the creation of the epileptic syndrome. At the present it is well known that an entire class of closed glands, and some others which functionate as open glands, exist. Their function, or at least the result of their functioning, throws into the organism substances, which, by themselves, would have a distinct action or which neutralize the toxic effect of certain poisons resulting from the life of the organism. Are all physiological, clinical and therapeutic experiments which have served to build up this theory solid and conclusive? This question is too long to discuss, but we have the facts and by classification let us try to find a practical explanation for them.

These reflexions on general pathology may at first appear misplaced in a paper such as this one, but I nevertheless would like to consider them briefly. The glands having an internal secretion, or better still, organs possessing a modifying action on the nervous system from the products which appear to result from their normal or pathologic function are, in the first place, the thyroid, parathyroid, suprarenal glands and hypophysis. The suprarenals have nothing epileptogenous in their history so they need not be considered.

The hypophysis is the most interesting from our viewpoint. Mairet and Bosc in their experiments on animals and man in a normal state and epileptic, have come to the following conclusions. When

a healthy man ingests the tissue of the pituitary body this is followed by a slight rise in temperature, weakness, gastro-intestinal disturbances and emaciation. When absorption takes place by means of intravenous injections a serious toxic state develops, similar to that resulting from intoxication by blood-serum and is only to be differentiated from the former by more marked myosis.

In the epileptic, intravenous injection or ingestion of the hypophysis *increases the number of attacks*. When taken by mouth it seems more particularly to cause paroxysms of delirium 3 or 4 days after. These paroxysms, which are equivalent to seizures, may completely resemble those that the patient ordinarily had, but it also appears that they are dissimilar.

The effect of the *artificial* introduction of the substance of the hypophysis into the body is very distinct but as no new facts have been published no legitimate conclusion can be, for the time being, formulated.

I shall not refer to the innumerable questions to which the thyroid gland has given rise, both in a normal and pathologic state. I would here simply refer to Case I of this paper. In this case a senile epilepsy was observed to give way to thyroid ootherapy with rapidity and effectively, to so marked a degree that no doubt is possible that it was the absorption of the thyroid gland and extract which caused the epileptic seizures to cease. Now, this patient was affected by an unquestioned thyroid insufficiency which goes to show that the primary cause of the epileptic attacks were the result of this insufficiency, and also because treatment seemed to bear out this view.

Now, what was the cause of the thyroid insufficiency? No phenomenon explains it better than the advanced degree of sclerosis of all the viscera which was noted in the patient and which is also the cause of the senility. For that matter, subjects which we commonly call sclerotics commenced their process in the vascular system and then by the progress of the lesion the entire organism becomes the seat of the lesions. And thus it is that the sclerosis of the endocrine glands may become the cause of senile epilepsy, at least if one may judge from some recorded cases and the therapeutic results obtained.

And these considerations lead me to the question of treatment,

which can be quickly disposed of. Symptomatic treatment by drugs to calm the medullary reflex irritability, such as bromides, etc., need not be considered, for they all are too well known.

I would merely remark that as far as exointoxications are concerned, the first measure to resort to is to eliminate the poison and then deal with the far-reaching effects which continue to show themselves after the primary cause has been eliminated. Here we will find scleroses of exotoxic origin, which brings us to the endointoxications, since total sclerosis produces an endointoxication and in elderly subjects it may readily create the epileptic syndrome.

It is clear that all these intoxications are distinctly exogenous at the beginning and the autogenous later on combine, so as to finally result in the production of a generalized sclerotic process.

For this reason opotherapy naturally finds its place among other forms of treatment. Opotherapy is both etiological and symptomatic, since it is directed against the functional insufficiency of certain endo-secretory organs. It is also symptomatic because it acts, not on the polysclerosis which is the basis of the pathologic process, but on the insufficiency which is symptomatic of the polysclerosis.

# Ophthalmology

---

## LATE POST-TRAUMATIC ATROPHY OF THE OPTIC NERVE

By E. MARGOLINE, M.D.

Geneva, Switzerland

---

MEDICAL literature is certainly not lacking in articles concerning the interesting question of the effects of traumatism of the optic nerve. Many theoretical, clinical and experimental researches have in recent years clearly proven that a two-fold mechanism should be incriminated in the action of trauma on the functional nerve of vision and that according to the case, the resulting visual disturbances are either due to direct mechanical action, such as a tear or complete section of the nerve by the traumatic agent itself, or to indirect lesions, such as fracture, hemorrhage, etc.

Now, the results of indirect lesions may appear only after a considerable lapse of time after the injury has been received and many oculists, who have studied the subject, have referred to this fact but only in a casual way without giving any details or according it the interest it deserves. It is known that it can happen and that is about all and the only particular mention that has been made is by Ywert in his book, "*Traité pratique et clinique des blessures de l'oeil*," published as far back as 1880, in which he has collected some cases scattered throughout medical literature. Other than this work, I have been unable to find any detailed description of this important ophthalmic subject, important both for the practitioner and specialist in forensic medicine.

I propose to study in this paper late post-traumatic atrophy of the optic nerve in as much detail as is requisite for the physician and shall report some unpublished cases from the records of Prof. Haltenhoff's clinic, likewise some instances that I have been able to collect from medical literature. I shall also refer to some theoretical points discussed by some writers in order to bring the question up to date.

Papillary atrophy of post-traumatic origin with functional disturbances coming on later, being intimately connected with direct lesions of the optic nerve in general, it is by the history of the latter subject that I shall begin.

Amblyopia and amaurosis following upon wounds and trauma inflicted over the orbit already attracted the attention of the ancient physicians, and it is almost traditional, when speaking of their interpretation, to quote the following lines from Littré's translation of Hippocrates: "Wounds received on the eye-brow and a little above it, obscure the sight; the more recent the injury, the less is the sight disturbed; but it often happens that it is lost as the cicatrix becomes older." Did Hippocrates intend to convey the idea that sight may be preserved immediately after the injury and become involved later on, "as the cicatrix becomes older"?

Again it was Hippocrates and later on Morgagni, Vicq-d'Azyr, Dupuytren, Ribes and others, who studied the pathogenecies of post-traumatic cecity and many theories have been proposed. For example, lesions of the brain and meninges extending to the nerve, commotion of the optic nerve and retina, the reflex action from a lesion of the trigeminal nerve, have all been incriminated in turn, not overlooking indirect fractures of the skull with bony callus, hemorrhage and contusions of the optic nerve as a result. Of all the theories, that of reflex amaurosis is the only one that was generally accepted, but it is also the one that had the most unhappy fate because the progress of science caused it to be completely abandoned.

At present, it is generally admitted that no matter what may be the immediate cause of atrophy of the optic nerve, it is most commonly fracture of the base of the skull which is at the bottom of them all; it is to this fracture that modern ophthalmology attributes the principal part in post-traumatic degeneration of the optic nerve.

The first reported case belongs to Chassaiguac who, in 1842, published the observation of an old blind man. The cecity being the result of the parietal bone and the autopsy revealed a fracture of the sphenoid compressing both the optic nerves. But the cases observed were very few, so that the article by Chassaiguac did not receive the attention it deserved,<sup>1</sup> so that Zander and Geissler in their work

---

<sup>1</sup> Chassaiguac. Thesis, Paris, 1842.

merely mention the very rare cases of lesions of the optic nerve from fracture of the sella turcica or fracture of the small wing of the sphenoid. It was only in 1872 that Galezowski spoke in precise terms of the frequency of traumatic amaurosis, assigning them a secondary place with a percentage of 18, the first place being accorded by him to tabetic amaurosis. As an immediate cause Galezowski even invoked a lesion of the nerve, the consequence of a bone lesion and a few years later (1879) he had a brilliant confirmation of his ideas in the writings of Berlin and Von Hoelder, who showed the extreme frequency of fracture of the vault of the orbit in fractures of the skull.

It was also Galezowski who gave at the same time some case reports of a late loss of vision and indicated the importance of the prognosis in cranial trauma. Vieuinne, in 1874, Hecquin at about the same time, also published examples, while in 1877 Richet, after having settled the much-discussed question of the existence of a callus in fracture of the cranial bones, speaks of it as one of the causes of late-appearing amaurosis.

Since this time, little has been added to this subject. In 1900, Mardellis,<sup>2</sup> and Ferron,<sup>3</sup> one year later, refer rather briefly to these cases. However, the publication of Ginestons,<sup>4</sup> in 1905, and that of Larayenne and Moreau, in 1907, should be mentioned. The latter writers, basing their theory on Nicod's thesis<sup>5</sup> on haematooma of the optic sheaths, invoke a latent papillary stasis in order to explain the ulterior appearances of disturbances of vision and record two cases in proof of this.

Late post-traumatic optic atrophy, since it belongs to the great class of indirect lesions of the second cranial pair should, I believe, bring up the following considerations:

(1) Indirect lesions of the second cranial pair follow fractures of the base of the skull. (2) Basal fractures of the skull are either direct or indirect; the former are exceptional, the latter represent the almost absolute majority of cases. (3) The nerve may be involved at any point of its tract—intracerebral, chiasma or peripheral portion—but it is more frequently in its peripheral portion, at the level of the

---

<sup>2</sup> Thesis, Lyons, 1900.

<sup>3</sup> Thesis, Lyons, 1901.

<sup>4</sup> Gazette hebdom. des sciences médicales de Bordeaux, 1905.

<sup>5</sup> Thesis, Paris, 1906.

optic canal, that it is injured, because it is at this point that the relations between the nerve and bone are particularly intimate and fractures frequent, as Berlin and Von Hoelder have shown.

Von Hoelder, out of a total of 126 fractures of the skull and all verified by autopsy, 88 fractures of the base of which 80 (90 per cent.) involved the orbit and 54 (61 per cent.) the optic canal. According to Pentscheff,<sup>6</sup> Prescott-Hewett found out of a total of 68 fractures of the base, 33 per cent. of fractures of the vault of the orbit, and the same writers refer to Schwartz's statistics of 102 basal fractures, 64 per cent. of which involved the anterior part of the skull and of these the majority may be regarded as involving the vault of the orbit.

(4) A fracture of the orbit may follow not only an injury received on the anterior portion of the skull, but also on its lateral aspect and occiput.

If we now examine traumatisms followed by late visual disturbances and take into consideration the various vulnerability agents, it will be seen that most usually it is a fall from a height, and of the 22 cases to be recorded further on, 11 are due to falls on the head. A blow from a stick, a stone, a heavy body falling on the head, a carriage or bicycle accident, etc., may also be the cause.

The gravity of the trauma is revealed by different symptoms, the most striking of which are, naturally, coma and delirium and rarely a deformity of the cranial bones. Usually it amounts to a simple loss of consciousness, nasal, buccal or auricular hemorrhage or hemorrhage from the orbit with palpebral or conjunctival ecchymosis with exophthalmus. In other cases oculomotor or facial paralysis or disturbances of the hearing, etc., are noted, so that out of a total of 22 cases only 4 are to be found in which none of the above symptoms were present. Therefore, I can only agree with Berlin, who, as far back as 1879, was positive that the trauma resulting in an atrophy of the optic nerve is always a severe one and I would add that this applies to traumatisms followed by late visual disturbances.

The frequency of these cases appears to me much greater than is generally believed, a fact easily explained, when it is recalled that the functional disturbances often come on only after a long lapse of time and their causal factor is all the more readily overlooked because

---

<sup>6</sup> Thesis, Geneva, 1895.

the trauma itself caused only slight and temporary immediate symptoms, sometimes these were even absent, although this is uncommon.

I have also compiled statistics of 365 cases of papillary atrophy observed in Prof. Haltenhoff's clinic and in 44 the lesion resulted unquestionably from a trauma. Of these 44 traumatic atrophies I found: (1) Direct lesions of the nerve, 12 cases = 27 per cent. (2) Indirect lesions with immediate loss of sight, 19 cases = 43 per cent. (3) Indirect lesions with late loss of vision, 8 cases = 15 per cent. (4) There remain 5 cases whose classification could not be established.

It is, consequently, evident that by their frequency, these cases are of considerable importance and the physician should be familiar with them and should know the consequences which may later on result from traumatism to the head, even if this seems to be slight. As Yvert very judiciously remarks: "One should always be very reserved in every case in which one is called to give an opinion on the probable outcome of such cranial traumatisms, because it is assuredly better to have the patient and his family aware of possible complications, although in many instances they are hypothetical, than to leave him a possession of a false security."

#### CASE REPORTS

**CASE I.**—(Clinic Haltenhoff. Reg. Cab. No. 1395.) S. B., 38 years old, received in May, 1874, a blow on the head without any serious trouble following. Four weeks later, the patient noticed that the vision had decreased; no pain or other symptoms. In January, 1875, the visual acuity was very weak on the right. He can distinguish fingers at only 70 centimeters distance. The supero-internal portion of the right visual field is completely wanting. Perception of red and green is lost, but blue is recognized normally. The left eye is also involved, its visual acuity being equal to 1/10.

**Diagnosis.**—Probable fracture of the orbit; progressive atrophy of the optic nerve.

**CASE II.**—(Clinic Haltenhoff. Reg. Cab. No. 9405.) Miss F., 18 years of age, while playing received a blow from a stick on the head. Following this trauma, there was a cicatrix to the right of the vertex. There was a hemorrhage from the wound at the time of the accident and pain in the head for one day, but other than this no ill effects were observed. About six months after the receipt of the trauma frequent epistaxis occurred and also nausea and frontal headache. At the same time frequent attacks of cloudy vision took place, sometimes before the right eye, at others before the left. Two years after the injury the patient no longer could see the upper portion of objects.

She then came to the clinic, when a distinct left superior hemianopsia was diagnosed by Prof. Haltenhoff, which completely corresponded with the sub-

atrophic aspect of the lower half of the left papilla. The right papilla and upper half of the left papilla showed nothing abnormal. Color perception was normal on both sides. This atrophy of the lower half of the section of the nerve trunk to the formation of an exudate (periostitis? meningitis?).

*Diagnosis.*—Upper hemianopsia, partial atrophy of the papilla, probably the result of an exudate.

**CASE III.**—(Clinic Haltenhoff. Reg. Cl. No. 1071.) B. I., 55 years of age, fell from a cart at the age of 45 years. Ten years later he came to the clinic complaining of visual disturbances, which had come on during the last eighteen months, and violent supra-orbital headache.

Antecedents excellent, no former disease, does not drink or smoke. After the accident there was no ecchymosis and no sudden loss of sight was noted.

The vision of the left eye is *nil*. The left pupil is dilated and does not react, but possesses an energetic consensual reaction. By ophthalmoscopic examination the left papilla is atrophied and slightly excavated; the vessels form a slight bend. The right eye is absolutely normal.

This case, like the preceding one, may be considered as being doubtful on account of the long interval existing between the trauma and the amblyopia. Therefore, it is recorded with all necessary reserve.

*Diagnosis.*—Fracture of the orbit, left-sided optic atrophy and cecity.

**CASE IV.**—(Clinic Haltenhoff. No. 1431. R. Cl. 1894.) C. F., 61 years of age, received a serious trauma thirteen years ago, on the right side of the forehead, the vision remained good. For the last four months it has become cloudy. No syphilis, no tabetic symptoms, no other disease.

The right eye can distinguish fingers at the distance of only one meter; the pupil is dilated, no reaction to light. The papilla is atrophied and allows one to see the outline of the cribriform lamina.

*Diagnosis.*—Right amblyopia; atrophy of the corresponding optic nerve, the result of an old traumatism.

**CASE V.**—(Clinic Haltenhoff. No. 14512.) M. D., 66 years of age, came to the clinic for a cataract. The left eye is completely blind; the papilla is atrophied and excavated; the blood vessels are thin. At the age of 9 years, the patient received a horse kick on the root of the nose and the left great angle of the orbit. Some time after the injury, vision of the left eye progressively weakened and was finally lost.

*Diagnosis.*—Post-traumatic atrophy of the optic nerve.

**CASE VI.**—(Clinic Haltenhoff. No. 17049.) A little girl 11 years old struck her head against a meal barrel; she did not lose consciousness but spat and vomited blood for three days. The accident occurred in the autumn and the following summer an outward deviation of the right eye was noticed. Prof. Haltenhoff found a complete cecity on the right, the consensual reaction persists. The papilla is atrophied and slightly excavated; the shadow of the cribriform lamina can be seen.

*Diagnosis.*—Trauma of the temporal region, right sided cecity, atrophy of the optic nerve.

**CASE VII.**—(R. Polyclinic No. 195.) C. J., 23 years of age, applied for treatment for visual disturbances of the left eye, which had become manifest during the past few months, without pain or other symptoms. Visual acuity is weak. The nasal visual field offers two notches, a supero-intern and infero-intern. Colors

are recognized at 2½ meters, excepting green. Reaction to light is very weak. The papilla is atrophied, with distinct limits; the vessels are thin.

Five months later the sight became still weaker and since he had formerly lost the fellow eye, the patient became completely blind.

He had never had any other affection and had always been well. No history of syphilis. Patellar reflexes normal, no tabetic pains. Romberg's sign absent.

Five years ago, the patient sustained a severe fall from his bicycle, striking on the left side of the head which resulted in a wound. At the time he remained unconscious for five days.

*Diagnosis.*—Fall on head from bicycle, amaurosis of left eye, atrophy of papilla.

**CASE VIII.**—(R. Cab. No 19614.) P. J., 43 years of age, fell from a height of 15 meters, sustaining multiple fractures, among them a fracture of the right orbital border. The right eyelid could be raised one week after the accident and the functions of the eye were still good. It was only after six weeks that the vision became cloudy and the lower field of vision disappeared. There was no diplopia and no pain.

One year after the fall, the right eye could not distinguish the fingers beyond the distance of 40 centimeters. The pupil was normal. The papilla was atrophied and excavated, permitting the shadow of the cribriform lamina to be seen.

*Diagnosis.*—Fracture of the orbit, atrophy of the right optic nerve, lower hemianopsia.

The following cases have been collected from the literature:

**CASE IX.**—(Heckin.) A workman was struck by a pile of sacks which fell from the height of 4 meters. Loss of consciousness, superficial wound of the external aspect of the right eyebrow.

At the beginning, vision was preserved, but after a few days it became cloudy and was completely lost seven months later. The patient suffered continuously from headache and periorbital itching which cleared after cecity occurred. Examination 18 months after receipt of the injury showed an atrophy of the right papilla. The left papilla is normal although the patient complains of diminished vision and headache on the left side.

**CASE X.**—(Hutchinson.) W. S., 28 years of age. Four years ago received a blow on the head from a leaded cane. Two years ago vision was still perfect, when suddenly a rapidly developing cecity took place in the right eye. Five days later there was sudden cecity of the left eye. At the same time there were violent orbital pains. The papilla appeared atrophied and excavated. Arteries thin, veins normal.

One month later the pains recurred and the patient at the same time suffered from incontinence of urine and faeces; epileptiform paroxysms also occurred, likewise an incomplete left-sided paralysis.

Autopsy showed a softening of the brain; the bulbæ, optic nerve and adjacent structures were adherent to each other; the optic nerve was very much softened. At the upper part of the brain the membranes were healthy.

**CASE XI.**—(Munchoff.) Male, 33 years of age, received a severe blow over the right occipital region, by being hit by a heavy door. At the time of the injury there was cloudy vision of the right eye, but other than this, there was no further trouble and the patient felt perfectly well.

A year and a half later, the patient noticed a weakness of vision in the right eye, followed two and a half years after receipt of the injury by a weakness of vision on the left and sometime afterwards by a diminution of the hearing on the left and pain in the legs. The visual acuity on the left is reduced at this time to one-quarter; the right eye is almost blind and has only retained the sensation of light. Both papillæ are atrophied, particularly on the right. The reporter of this case considers that it is a probable lesion of the brain.

**CASE XII.**—(Galezowski.) M. D., 31 years of age. Atrophy of the left papilla, edema of the right papilla. One month before cecity occurred he had sustained a fall on the head and remained unconscious for two days. The vision began to weaken two weeks after the injury.

**CASE XIII.**—(Ory.) Abscess of the orbit following a fall on the head five days before. The vision, which was at first intact, disappeared on the fourth day. Forty days later there was partial atrophy of the papilla. The writer supposes that there was probably a fracture of the orbit and extension of the inflammation to the tissues surrounding the nerve itself.

**CASE XIV.**—(Vieusse.) Fall, followed by loss of consciousness. Contused wound of the orbital arch. Subconjunctival and palpebral ecchymoses. Two weeks later, vision on the right was cloudy but in ten days it had completely disappeared. The ophthalmoscope showed an atrophied papilla with clear cut limits; the vessels are not reduced in size.

**CASE XV.**—(Yvert.) A woman fell from the first story of a building. The immediate result was not serious and the patient did not even become unconscious. However, at the end of a few days, she fell ill, had headache and vomiting. Six weeks after the accident, the patient noticed a cloud before the left eye and three weeks later the same phenomenon occurred on the right. Two years after the accident she was almost blind. The ophthalmoscope revealed a simple progressive bilateral atrophy of the optic nerve.

**CASE XVI.**—(Mardellis.) Patient received a blow from a bottle over the right orbital arch. Nasal and auricular hemorrhage followed; there was no loss of consciousness or visual phenomena. Six weeks after the injury, there was weakening of the sight on the right. Four months after the accident the right eye became almost completely blind. The patient can see the fingers at only 10 centimeters distant. The papilla is a trophied and excavated; the vessels are normal.

**CASE XVII.**—(Hoffmann.) H. F., 32 years old, fell from the height of 2 meters. He had a nasal and buccal hemorrhage and remained unconscious for thirty-six hours. Vision remained good for five weeks and then began to be cloudy. He came to the clinic six weeks after the accident and an absolute cecity was found on the right and a temporal hemianopsia on the left. Both papillæ were atrophied, this being more marked on the right.

**CASE XVIII.**—(Hoffmann.) W. J. fell on his head from a height of 2½ meters and sustained a fracture. The vision became weak three weeks later. Six months after the injury he came to the clinic with complete amaurosis of the right eye and a corresponding atrophy of the papilla.

**CASE XIX.**—(Ginestous.) Male, 30 years of age, had a carriage accident, following which there were no immediate results excepting a slight palpebral ecchymosis. A week after the accident there was a sudden loss of vision on the

left. Two years later there was complete loss of vision on this side. The diameter of the papilla was normal with distinctly outlined borders. The papilla is white.

Case XX.—(Dufour and Gonin.) A child, 11 years old, had a fall in November, after which there was no loss of consciousness, but frequent headaches occurred and even some lowering of the intelligence. From January of the next year, a marked weakening of the vision was noted and when examined in February, the child could only count the fingers at a distance of one meter. The left visual field was reduced externally, the right visual field was reduced to its nasal half (temporal hemianopsia).

By the ophthalmoscope, both papillæ were pale but the vessels were not diminished in size.

During the following months the vision continued to weaken and in July a complete right cecity was noted and a distinct left hemianopsia. Two years later the vision of the left eye was reduced to the sensation of light. Both papillæ were totally atrophied.

Case XXI.—(Laroyenne and Moreau.) G. I., 20 years of age, sustained a violent fall on the head, resulting in an extensive wound over the right parieto-temporal area; epistaxis from right nostril, ecchymosis over the temple and in eyelid; coma, followed by violent delirium. Consciousness returned in a week and the patient did not present any motor or sensory disturbances. Twenty days later the sight began to diminish. Nothing to be noted externally excepting a sluggish pupil. Right papilla red and somewhat tumefied, vessels normal. Visual field greatly diminished.

Twenty days still later, the left eye is also involved, its visual field is narrowed, sight being one-eighth. The left papilla is red with irregular borders, and atrophied in its temporal half.

Four months after the injury, there is a total post-neuritic atrophy of the right optic nerve; the left papilla presents the same changes already noted. There was probably a transverse fracture of the base of the skull involving both optic canals.

Case XXII.—(Larayenne and Moreau.) B., 60 years of age, hit his head against a stone. Extensive scalp wound and epistaxis but no further symptoms. Nine months later the vision became weakened. The patient applied for treatment nine months after the injury. Left eye absolutely blind, the pupil is dilated and the reflex to light abolished. By the ophthalmoscope, papilla white with indistinct borders. Arteries occluded, veins tortuous, with two characteristic bandlets.

Probably there had been a compression of the optic nerve from fracture of the optic canal; a papillary stasis resulted, followed by atrophy.

Galezowski says that "the genesis of traumatic neuro-retinitis is very varied and it would be in contradiction with clinical facts to endeavor to explain all cases by a single theory." There do exist several theories which may all be admitted, but no one of which corresponds to late cases of amblyopia. A splinter of bone, a callus, a simple contusion, a haematoma, a meningitis or a periostitis may all give rise to atrophy of the optic nerve, but no one of these factors is

inevitably necessary in each case, and according to the symptoms one or the other will present itself to the mind of the physician.

According to the type of atrophy to which they give rise, I shall divide the factors into two categories, namely, (1) Those resulting in simple atrophy of the nerve, and (2) those which, before the atrophy, have given rise to inflammation and papillary stasis so that the two last-named processes represent the direct causal factor of the degeneration of the nerve.

*Simple Atrophy.*—(a) Bone callus. As Ferron has properly remarked, the hypothesis of a callus is the first to come to one's mind, because it is most natural to search for secondary complications in order to interpret the late phenomena. Now, compression by a callus is clearly a complication which itself arises tardily because the formation of a callus requires a certain time. It would seem that it is only from lack of post-mortems that this hypothesis has not been proved, because this supposition, from its very simplicity, has not raised any adverse criticism.

But there is a very serious objection to it. The bony callus formed at the base of the skull is, in the larger number of cases, of no considerable size and the proportion of cases of exuberant callus does not correspond to that, much greater, of post-traumatic amaurosis which could be thus explained. I, therefore, must admit that although this causal factor is probably real in a certain number of late post-traumatic optic nerve atrophies, it is altogether insufficient to explain all the cases.

(b) *Inflammation of the neighboring structures* to the optic nerve may involve the latter. For example, a meningitis may extend to the sheath and then to the nerve itself. Vision may be all the later in diminishing, the later the development of the basal meningitis. According to Ferron, the phenomena of periostitis must be mentioned, this process which so often occurs in detachment of the periosteum. I will readily admit this possibility, but unfortunately I have been unable to find any unquestionable example in the literature, and Ferron himself does not offer any case in proof of this.

In order that all these suppositions shall not remain mere hypotheses, the proof of necropsies is absolutely necessary, and it is much

to be desired that in appropriate cases which come to autopsy, attention should be paid to both basal and meningeal lesions.

An abscess of the orbit occurring after a trauma may extend to the optic nerve, producing a retrobulbar neuritis and an atrophy of the papilla as an ultimate consequence, and the case reported by Ory is distinctly proof of this.

*Contusion of the Optic Nerve.*—It is by this theory that Damon and Pentschiff explain late post-traumatic optic nerve atrophy. The latter writer maintains that the shock necessary for the production of a fracture of the very mildest type and the hemorrhage which results, causes a compression of the nerve and this is followed by its degeneration. Such may, perhaps, be the clue to certain cases of the so-called essential papillary atrophy, which really are due to an unrecorded trauma.

*Atrophy From Neuritis and Papillary Stasis.*—Let me at once say that this variety is much less commonly met with than simple atrophy, for the reason that papillary stasis does not of necessity lead to ultimate atrophy and also because, and this is of still greater import, traumatic neuro-retinitis is itself an uncommon lesion. Out of a total of 25 cases of neuritis of the optic nerve, Galezowski only found two having a trauma as a causal factor, while according to Hulke, the proportion is still less as he found only two instances of trauma out of a total 39 cases of neuritis of the optic nerve.

This, however, does not prevent one from invoking several causal factors for the purpose of explaining the numerous varieties of traumatic neuro-retinitides. Occasionally, as in simple atrophy, the cause is a traumatic meningitis, in other cases it is an intracranial collection of blood which infiltrates the sheaths of the optic nerves. Finally, it may also be an infiltration of the sheath, but having a peripheral origin, and in these cases the haematoma of the sheath is the result of rupture of the latter and the vessels belonging to it, or it may be the result of rupture of the central retinal vessels before they enter the nerve. A papillary stasis may result from compression of the vessels from a callus, splinter of bone, etc.

Now, how can the later disturbances of the vision be explained? It is, I think, possible to do so from their etiology, because in certain conditions, as in meningitis, for example, the papillitis may appear

after a variable interval of time. But another fact well explains the visual integrity, even when there is a papillitis.

It is known that a papillary stasis may exist for a very considerable length of time before it results in changes in the vision. Panas has recorded a very interesting case without visual disturbances and without changes in the nerve fibres at the end of a year. Dufour and Gonin have also called attention to the fact that a papillary stasis may, for months, only cause temporary cloudiness or obscuration. The central and peripheral field of vision remain free for months, but disturbances of visual acuity, when once they begin, assume a rapid course and soon terminate in cecity.

From all this it follows that, in certain cases an uninterrupted chain can be discovered between traumatisms and very late occurring optic nerve atrophy, if an ophthalmoscopic examination were made immediately after the receipt of the injury and as Laroyenne and Moreau have said, "It is essential that every person who is suspected of having a cranial lesion should be examined as to the condition of the eyes, as soon as possible after the receipt of the injury. One should not wait until the patient complains of visual trouble, because we know that these are not always related to papillary lesions."

If we now compare all these different interpretations, it will be seen that in spite of their number, a point remains which is completely obscure. In fact, how are we to explain the simple late atrophy, without oedema of the papilla, without symptoms of meningitis and which reveals itself only after the lapse of one or more years? A callus or a contusion of the nerve would have given rise to symptoms of compression a long time before; an inflammation of the surrounding structures would do likewise with the symptoms belonging to each. Unfortunately, I have no explanation to offer in spite of the uncontested existence of such cases.

The pathognomonic symptom of all our cases is unquestionably an absolute integrity of the vision immediately after the receipt of the injury. The only phenomena to be noted are the luminous sensations and dazzling of sight experienced by the patient. The latter phenomenon follows irritation of the optic nerve and may continue for several days with some intensity, but taken by themselves, each is of short duration. Visual acuity remains normal for a more or less

long time, varying according to the circumstances. Frequently the lowering of sight appears after a few days, while in other cases, some weeks, months or even years pass before this takes place. In two of my personal cases here reported, the degeneration of the optic nerve could not be accounted for by any other causal factor than an old trauma, dating back ten years in one case and thirteen years in another. It is evident that this far off genesis is always very hypothetical and should only be admitted under the strictest reserve.

Most frequently only one eye is involved, but sometimes both are almost at the same time. Again, others present a very appreciable interval between the time of changes taking place in each eye.

Visual weakness is accompanied by a narrowing of the visual field. In cases of lesions of the chiasma or the optic bandlets, a nasal or temporal hemianopsia occurs. If the lesion is nearer to the periphery, the reduction of the field of vision is usually concentric, but a notch or a central scotoma are not contra-indications to the diagnosis. As to color perception, red and green are the first to disappear.

From all these symptoms an optic nerve atrophy may be suspected *a priori*, even should the ophthalmoscope show nothing, because sooner or later it will almost always reveal a white, decolorized papilla, sometimes excavated, presenting sharp, distinct limits, allowing the cribiform lamina to be seen in outline. The blood-vessels may remain normal for a long time and might be supposed to be a good diagnostic sign, but they always end by narrowing.

Such is the aspect of the papilla which has undergone simple atrophy. But the same is not true of the more infrequent cases of atrophy of the optic nerve resulting from a papillary stasis, whose preëxistence may be demonstrated by the ragged and irregular contours of the papilla and by its cloudy aspect which prevents one from seeing the outline of the cribiform lamina. The vessels are narrowed, but at the beginning of the atrophic process the veins may still retain the tortuous aspect observed in papillitis. Finally a deposit of pigment around the atrophied papilla testifies to a former inflammatory process.

These changes having been noted, the diagnosis of post-traumatic disturbances having been established, what should the prognosis be? Personally I believe that it is always very serious because if the

cases here recorded are consulted it becomes clear that there is always a progressive loss of sight. In the majority of cases an absolute cecity occurs and this usually very soon after the appearance of the subjective symptoms. The prognosis is made still worse in the beginning from the uncertainty of what may happen in the fellow organ, which may later on become involved.

This unfavorable prognosis greatly increases the importance of these cases from the forensic viewpoint. Take, for example, a laborer who is perfectly strong and healthy, without any history of past illness or syphilis and without any hereditary taint, and let us suppose that during his work his skull is struck by machinery, or otherwise. The accident does not produce any immediate untoward result and appears to be unimportant. But later the subject complains of disturbances of vision; an ophthalmoscopic examination reveals a more or less pronounced atrophy of the papilla and since there are no other causal factors to explain the process in the fundus, one is obliged to attribute the visual diminution to the cranial trauma received sometime before. The subject has become incapable to work and so can exact a large indemnity, because it is an instance of employer's liability.

Another question of forensic interest may come up, namely, that of the responsibility of a person who has employed violence to another. This responsibility increases from the moment visual disturbances appear and atrophy of the optic nerve becomes declared. It is all the more serious since, as I have tried to show, the eye is bound to cecity, and sometimes both organs. Therefore, I cannot insist too strongly on the medico-legal aspect of these cases. Unfortunately the question is complicated because the diagnosis must be one of exclusion. The record of the trauma is insufficient, and one must eliminate all other factors which can produce atrophy of the optic nerve, which is not always an easy task.

# Rhinology

---

## SINUITIS AND HEADACHE—TREATMENT

By LEWIS FISHER, M. D.

Philadelphia

---

DISEASE of the nasal accessory sinuses, in either the acute or chronic form, is probably the most frequent affection of the upper respiratory tract. Most of the so-called catarrhs, nasal discharges, or cases of post-nasal dropping producing constant hawking and spitting, as well as the more or less temporary obstructions to breathing, are probably traceable to a chronic infection within the nasal accessory sinuses. Either type of infection may manifest itself in various degrees of severity: viz.

1. A purely local disturbance limited to the sinus affected, with pain, swelling, nasal obstruction, etc.

2. There may also be present systemic effects. These may range from the violent toxæmias with fever and marked general prostration to the lower grades of toxic absorption with various effects in remote organs—the sinus condition acting as a source of focal infection.

3. These may result in involvement by extension of the neighboring organs such as the eye, meninges, etc.

The scope of this paper will not permit a lengthy discussion of the symptomatology and pathology of these various conditions. There is one symptom, however, that is present in practically all forms of sinuitis, namely headache, and we wish to devote our attention to a discussion of this symptom. In many instances of "headache" there is a definite history of nasal obstruction with a discharge of pus from one or both nostrils—in fact, these are so evidently and patently "sinus cases" that even the patient himself recognizes this. There are, however, a number of cases where the cause of the headache is rather obscure. To the casual observer, or even to the rhinologist, no *apparent* nasal condition is present to account for the headache.

Nevertheless, a more careful search would show that the seat of the trouble in most of them is within the nose or accessory sinuses.

Headache as we understand it must be an irritation or affection of the chief sensory nerves of the head—the fifth pair of cranial nerves. The various subdivisions and ramifications of this pair of nerves supply with sensation practically the entire head, including the meninges. Before one can be aware of that conscious sensation we call headache, it would seem that the sensory distribution of the head must be affected by the pathological process responsible for the headache. The disturbance in question may be found directly along the fibres of the fifth nerves themselves, or, on the other hand, it may be situated remotely and affect them through the medium of the circulation, or they may be affected reflexly. This conception of the causation of headache must not be misunderstood. Every clinician knows that eyestrain is a prolific source of headache and is readily relieved in those cases by proper glasses. It is also a matter of common experience that headache is frequently present in many toxic conditions, particularly those brought about by constipation; that it is also present in many pelvic disorders, etc. Headache is, nevertheless, always in the head, and not in the bowels or uterus or in the eye. Before any of these conditions could produce headache, it is essential that they affect directly the fifth or sensory nerve distribution. The rich connection of the trigeminal nerves with the sympathetic and central nervous system generally makes this possible. In the case of headache due to eye-strain they are affected reflexly by way of the oculo-motors, whereas in the case of the headache due to constipation or some pelvic disorder, the fifth pair of nerves are affected either through the circulation or reflexly or both. There is abundant evidence to convince even the most skeptical that peripheral nerve irritation is capable of producing severe headache. Most clinicians have seen numerous instances of headache that can be instantly relieved by shrinking up the mucous membranes of the nose or the turbinate bones. There, the headaches are undoubtedly due to pressure of the turbinate bones against the nasal mucosa, in other words, a peripheral irritation of the fifth nerve distribution. A local application of adrenalin chloride or cocaine shrinks the mucous membrane and turbinates, thereby relieving the pressure on the peripheral nerve filaments and causes the headache to disappear. A sensory nerve

need not, however, be irritated or affected peripherally in order to produce sensory phenomena. There is no doubt that a good many toxæmias produce headache by affecting the fifth nerves centrally. In this way we can explain those toxæmias in which headache is not the only symptom but is associated with other sensory manifestations. In the case of the "dizzy" headache it is probable that the toxæmia affected not only the fifth nerve distribution but also the vestibular apparatus. Where the headache is associated with vomiting, the toxæmia has also affected the phrenic nerve and the tenth cranial nerve. The very fact, however, that in the low grades of intoxication, headache is practically the only symptom present, would strongly suggest that in these toxæmias it is the peripheral portion of the nerve distribution that is most frequently affected. The best example of a toxæmia of this type is the one resulting from constipation. Headache is a very prominent symptom of this condition. Assuming that the constipation produces a toxæmia which circulates in the blood, is it not rather surprising that headache is the only symptom? The blood supplies simultaneously many other sensory nerves, including all the nerves in relation with our special senses. One would expect that the toxæmia would irritate the eighth nerve distribution and cause dizziness or affect the optic nerve distribution with resulting visual disturbances, or that there would be sensory phenomena generally with pain in the various parts of the body. Why should this toxæmia affect particularly the sensory nerve supply of the head in the form of a headache? The answer is probably this—the toxic condition affects most frequently *that* sensory nerve which has the most vulnerable distribution. Within the nasal cavities and their associated accessory sinuses there is a very large area of mucous membrane richly supplied with sensory nerves. Conditions of life in a modern industrial centre aided by the many climatic changes to which one is subjected predispose the nasal mucous membranes as well as the membrane lining the nasal accessory sinuses to all sorts of pathological changes. The nasal accessory sinuses, however, are particularly an easy prey for all forms of inflammation for the reason that the ostia or openings by which these cavities are in communication with the external world are not placed in the best position for drainage. It is known that in lower animals inflammation of the sinuses is rather rare. Walking on all fours as they do, the ostia

are so placed in relation to the accessory sinuses that they are located in the most dependent portions of those spaces—the most favorable position for drainage and ventilation. When the human animal assumed the erect posture, Nature for some reason failed to move those ostia, and left them in their original position, with the result that in the human being, they are placed considerably above the floors of these cavities.

Swelling of the turbinated bodies may very easily occlude those minute openings and cause an oedema and congestion with or without subsequent infection of the sinus contents, with the result that the fifth nerve distribution—so rich in those parts—suffers and manifests itself as headache. The swelling or turgescence of the turbinates may be the result of a local condition, but it would also be a simple matter for a toxæmia such as produced by constipation to produce a slight congestion or an interference with the normal elasticity of the turbinates with its usual sequela of poor ventilation and aeration, fifth nerve irritation and headache. In other words, it is probable that even in constipation-headache a nasal condition is the immediate cause.

The relation between sinuitis and headache may be summed up as follows:

1. Headache is produced by an irritation of the fifth cranial nerve distribution either peripheral or central, most frequently peripheral.
2. A pathologic process usually affects the most vulnerable portion of a structure. The most vulnerable portion of the fifth pair of cranial nerves is the portion found exposed to external influences in the large area of mucous membrane within the nasal chambers and their accessory sinuses. The fifth nerves are therefore affected most frequently in their periphery.
3. The nasal accessory sinuses because of their peculiar anatomic structure with poor means of ventilation and drainage are most frequently affected—hence, fifth nerve irritation, or headache, is such a common symptom in sinus inflammation.
4. Many headaches are undoubtedly not of nasal origin, but those directly traceable to a nasal condition are sufficiently common to suggest that the nasal cavities and their accessory sinuses be first searched in an effort to determine the cause of headache.

5. The diagnosis that the sinuses are the cause of the headache in any particular case can frequently be made only after treatment is instituted. That is, the prompt relief afforded, in spite of the absence of the usual signs of sinus inflammation is *ipso facto* proof of its cause.

The treatment of sinus infection whether acute or chronic must follow the same general surgical principles that infections follow elsewhere, namely, all efforts must be made toward establishing proper drainage. The first and most important procedure is directed toward reestablishing normal drainage through the various sinus openings. This is accomplished by making intranasal applications of cocaine or adrenalin chloride in the region of the ostia. In acute cases frequent applications with rest in bed and aspirin and atropin internally, combined with free catharsis, will usually relieve the patient. Should the fever, pain and other symptoms continue, showing that the efforts to keep the ostia patent are of no avail, surgical measures are indicated. These should be of the simplest kind. Usually high amputation of the anterior portion of the middle turbinate will suffice. This, however, is very rarely necessary if in addition to shrinking the middle turbinate region with cocaine and adrenalin one employs negative pressure or suction following each application.

The chronic forms of sinuitis are not so easily amenable to medical treatment, and surgical measures have to be resorted to more frequently. Here too, however, the best results are obtained from the conservative treatment of the case rather than the radical exenteration or curettage of all the sinuses. Where the disease has actually produced polypoid growth or caries of the bone, these of course must be removed. It is well, however, to sacrifice as few of the structures as possible—above all care must be taken to conserve the middle turbinated bones. The latter should never be removed unless they are *absolutely* diseased and are beyond any hope of regeneration. All forms of treatment have one object in view, namely, elimination of the purulent matter in order to give the mucous membrane a chance to regenerate. There is one form of treatment which is universally applicable in all cases of sinuitis, in so far as it accomplishes the very thing that is essential for a cure of this condition—emptying the sinuses of their purulent contents—treatment by “suction.” This has been found so efficacious and satisfactory that

since its universal employment in these cases, surgical intervention is but rarely necessary. One of the greatest difficulties in the treatment of all sinus infections is their chronicity. The peculiar anatomical configuration of the sinuses makes a direct approach to most of them through their natural openings almost impossible, and proper drainage is therefore most difficult to establish. In order to obtain success, treatment must be extended over a long period of time and many of the failures are the result of impatience and lack of co-operation on the part of the patient. The frequent visits to a specialist's office, entailing, as it must, considerable expense, makes it frequently impossible for the patient to persist long enough to obtain relief. At the present time there are many types of electrically operated machines easily adapted for treating the nasal cavities by suction. When treatment is started it is not essential that the patient come to the specialist's office for every treatment. This can be carried out daily if necessary by the family physician with an occasional visit to the rhinologist to supervise the progress of the case. The patient's nares are first sprayed or cleansed with an ordinary alkaline or normal salt solution, then the middle turbinate region is brushed over with a 10 per cent. solution of cocaine hydrochloride carried on a cotton-tipped applicator. After a lapse of two or three minutes—a sufficient time for the shrinking of the mucous membrane—suction is applied in the following manner: A small, rounded glass nozzle is tightly inserted into one nostril while the other nostril is closed with the finger. The patient is then told to swallow or to say the word "hock" the object being to raise the soft palate and shut off the upper respiratory tract from the mouth and trachea. The operation of the suction machine creates a negative pressure in all the spaces between the nares anteriorly and the naso-pharynx posteriorly. As soon as the suction becomes uncomfortable the finger is removed from the closed nostril and the negative pressure disappears. This treatment is kept up for several minutes, just as long as there is any secretion brought out. In this way we accomplish a two-fold purpose. The accessory sinuses, even the remotest ones, are emptied of their contents, while at the same time the negative pressure induces a hyperæmia or a mild passive congestion of the parts, altering their nutrition and favoring reparative processes.

The following case histories illustrate the type of cases where such treatment produces most gratifying results.

**CASE I.**—Mrs. R., age 63, was admitted to the Mt. Sinai Hospital on May 23, 1918, with chief complaint of frontal headache, peculiar sensations in the left hypochondriac region extending toward the epigastrium, epistaxis, marked constipation and persistent vomiting. The illness began four days before the patient's admission to the hospital. The first symptom noticed was a distressing headache accompanied by epigastric pain. She vomited several times in succession and just before admission to the hospital lost control of the bladder.

The family and past medical history were negative. Examination showed the patient to be greatly prostrated, temperature 103, respiration 32 and a pulse ranging between 90 and 100. The chest and abdomen were negative. Patient's constipation was overcome by the administration of castor oil and ox-gall.

The blood showed haemoglobin 85 per cent., red blood cells 5,000,000, a leucocytosis of 16,000.

Wassermann of the blood was negative.

The patient's condition remained about the same for five days. The temperature varying between 103 and 99—always higher in the evening. Respirations were about 30 and the pulse continued to range between 90 and 100. This patient, having been admitted to the hospital as an "abdominal case," did not have a nasal examination made until the fifth day after her admission. There was found at that time a chronic ethmoiditis with a tendency to atrophic rhinitis. The right nostril contained a large polyp but there was no evidence of any acute inflammatory process in the sinuses. The nares were cleansed, a 10 per cent. solution of cocaine applied to the middle turbinate region of both sides and moderate suction applied for several minutes. This was successful in bringing out a small quantity of pus. That night the temperature did not go up above 99½. The patient was treated in the same way for the week following—once every twenty-four hours. The headache disappeared after the first treatment; the general condition began to improve rapidly; the temperature came down and remained normal. The patient was up and out of bed in two days after the nasal treatment was first instituted and was discharged in her usual state of health five days later.

This case well illustrates the value of a careful investigation of the nose and sinuses in all cases of obscure headache due to some undetermined infection. This patient was admitted to the hospital with the diagnosis of intestinal obstruction. A short study of the case, however, convinced the clinician that he was dealing with some type of infection the source of which, however, could not be discovered. A thorough search of the lungs and abdominal organs as well as a careful study of the blood and urine failed to point to any focus of disease. It was only the distressing headache accompanied by protracted and marked drowsiness that directed suspicion toward the nose and the accessory sinuses.

**CASE II.**—Miss K., age 30, was taken ill on April 10, 1918, with malaise and slight fever. Two days later the symptoms became so aggravated that she was compelled to go to bed. At that time she appeared to have an asthmatic type of bronchitis with typical acute attacks of asthma from time to time. The temperature, 100° in the beginning of the illness, rose to 103 at times. Nasal examination showed the septum deviated to the right in its upper portion, but no evidence of any acute process in any of the nasal accessory sinuses. Shrinking of the middle turbinates with cocaine gave no relief. Patient was advised to have her nasal condition corrected as soon as the asthmatic attacks subsided. A rest in bed for a week combined with good nursing and medical care relieved the patient of her asthma and bronchitis, but in spite of the relief she was not getting well. Temperature continued and patient developed a sort of semi-stuporous condition. She would go off to sleep the moment she was allowed to remain alone. The temperature, although not high, was septic in type; the headache was excruciating. Patient was greatly prostrated and appeared to be very ill. Another examination of the nose failed to reveal anything new. At the suggestion of the attending physician the nasal accessory sinuses were treated on the basis of an acute infection—that is, in addition to shrinking up the ostia, suction was applied to both nares. There was immediate relief following the first treatment. The headache became less and temperature became lower. Under this treatment there was complete recovery in ten days.

**CASE III.**—Mrs. R., age 60, the mother of a physician, was suffering from cardiac disease for many years and subject to cardiac

asthmatic attacks lasting several months at a time. She gave a history of being subject from time to time during the past several years to acute attacks of headache, malaise and inexplicable fever which would be ushered in with a chill. These attacks would last several days and appeared to be uninfluenced by any type of treatment. During one of these attacks in the beginning of June, 1918, a nasal examination was made. The septum was found thickened in its upper portion, exerting pressure against both the right and left middle turbinated bones. The turbinates themselves were boggy and turgescent and appeared to be covered with a sticky semipurulent fluid. These were well shrunken by a solution of cocaine in adrenalin chloride and the nares thoroughly sprayed. This was followed by an immediate drop in temperature and a complete cessation of the excruciating headache which had lasted for several days prior. The following day, with the exception of an evening temperature of 100° the patient was in her usual health.

CASE IV.—Mrs. R. S., age 44, the mother of five children, has been a sufferer from "stomach trouble" for many years. There were many symptoms suggestive of a mild infection of the gall-bladder and its ducts. Her chief complaint was more or less constant headache. These attacks of headache would recur from time to time with greater or lesser severity until for a month or two before coming under observation her headaches were practically constant. Patient was so incapacitated because of the headaches that she was unable to attend to her household duties. Her medical attendant made a thorough search for any possible cause of the headache, including an X-ray examination of the head, teeth and eyes. The teeth were found to be abnormal and the eyes showed a refractive error; both of these conditions were corrected without relief from the headaches. A nasal examination showed a tendency to atrophic rhinitis, the turbinates were of moderate size, septum fairly straight. There was no evidence of purulent secretion in any portion of the nares. Irrigation of both antra and sphenoids showed no pus. There was no tenderness over any of the sinuses; transillumination was negative. The report to the family physician was that the nose was probably not the seat of the patient's trouble. At his urgent request the patient was examined again and at his suggestion was treated as a sinus case. The turbinates were shrunk with a 10 per cent. solution of cocaine and

suction applied to both nares. A small quantity of mucoid material was obtained. The relief was instantaneous. Bacteriologic examination of the mucopous obtained showed a culture of diphtheria bacillus. Treatment of the patient by suction and by injections of autogenous vaccine as well as diphtheritic serum quickly cleared up the case.

There is nothing startling or new about cases of accessory sinus infection; they have been known for many years to the more intelligent observing physician. These cases, however, are of double interest because a nasal examination at the time failed to show any evidence of sinus disease; and under the old standards of examination, namely, inspection, transillumination, irrigation and X-ray, the sinuses would have been pronounced as uninvolved. The use of the suction, however, has served in these cases not only as a diagnostic means but at the same time as a curative agent—all of these cases being promptly relieved by the application of the negative pressure to the nasal chambers. The ease with which suction can be applied and the convenient way in which these small portable machines are manufactured makes it feasible for every practitioner to use this means of diagnosis and treatment where help at the hands of a specialist is not available. These histories show that ordinary examination of the nose and throat is not sufficient in these obscure cases of headache and fever. These cases should be treated as sinus cases, all evidence to the contrary notwithstanding. The treatment is simple, easily carried out, is never harmful, and, on the other hand, will prove beneficial, and helpful in most instances. Even in cases where there is infection elsewhere in the body the distressing symptoms of headache and drowsiness will be relieved by taking care of the nasal accessory sinuses.

# Surgery

## ANÆSTHESIA IN EUROPE ON THE WESTERN BATTLE FRONT

By P. J. FLAGG

THE individual blessé who goes to make up the casualty list of the belligerents does not differ in his reactions to various anaesthetic agents from the ordinary routine hospital case with which we are familiar. The fact that he has been wounded in a "spad" ten thousand feet in the air or in a dugout on a hillside does not of itself constitute a serious problem for the anaesthetist. Indeed, the fact that these patients are practically limited to adult males who are organically sound simplifies the choice of the anaesthetic. In civil practice one is accustomed to seeing accident cases suffering from loss of blood and traumatic shock. The large urban hospital with an active ambulance service presents cases quite similar to those found in the Casualty Clearing Station.

The difficulties presented by military anaesthesia may best be studied by explaining first of all the relative position of Casualty Clearing Station, Base Hospital, etc., to the firing line. These positions are as follows:

Zone of Advance.....	{ First Aid Dressing Stations Field Hospital Divisional Ambulances Surgical Auto Unit (Autochir)
Intermediate Zone.....	{ Evacuation Hospital (French) Casualty Clearing Station (English)
Zone of the Interior.....	{ Base Hospitals Specialty Hospitals

The Zone of Advance is about seven miles broad. It occupies the territory immediately behind the firing line.

The First Aid Dressing Stations in the trenches and dugouts attend to urgent cases of hemorrhage, apply splints to fractured limbs, dress fresh wounds and give morphine hypodermically.

The Field Hospitals which are scattered about in convenient locations within this zone receive patients from the Dressing Stations. Practically all cases requiring immediate operation are done here, i.e., amputations, injuries of the head, neck and abdomen which cannot be deferred, etc.

Divisional Ambulances move from one Field Hospital to another or from the Field Hospitals to the Intermediate Zone.

Auto Surgical Units, or Autochir furnished with complete operating equipment, constitute movable Field Hospitals in themselves.

The Intermediate Zone extends from seven to fifty miles behind the firing line. It occupies a broad stretch of territory immediately behind the Zone of Advance.

The Evacuation Hospitals (French) or Casualty Clearing Stations (English) situated in the zone receive patients from the Zone of Advance. All cases which are too sick to be transferred to the Base Hospitals are operated upon here. In fact, the major portion of all primary operations are performed at this location. Shell fragments are removed, decompressions are accomplished, open reductions of fractures are done. Intra-abdominal explorations and intestinal anastomoses are performed. Briefly speaking the Casualty Clearing Station proposes to treat all cases requiring interference which have not been attended to by the Field Hospitals and which are too critical to await transportation to the Zone of the Interior.

The Zone of the Interior is that region lying about fifty miles behind the firing line.

The Base Hospitals, the first permanent medical establishments of the military system, are found here. Base Hospitals may be a long distance from the scene of conflict, for example witness The First London Base Hospital and Base Hospital No. 1, Bronx, New York. The Base Hospital is the ultimate destination of all cases who can await operation and who can endure the hardships of a long and tedious journey. The Base Hospital treats cases of ostitis, osteomyelitis, infected fractures, suppurating wounds, etc. The work of the Base Hospital closely resembles the ordinary civil surgery with which we are familiar, with the exception that a great many of the cases have already been operated upon.

The Specialty Hospital, as its name implies, is for the purpose of

giving attention to conditions which require special training; *i. e.*, brain and cord work, eye, ear, nose and throat, etc.

The Base Hospital and the Specialty Hospital is, so to speak, the court of last appeal for the surgical and medical problem in the Army and Navy.

Having briefly described the circumstances under which military anaesthesia is conducted, we may proceed to a closer inspection of the immediate difficulties presented by the blessé. These difficulties may be summed up as follows:

The lack of preliminary preparation.

The unusual and urgent need of a speedy induction and a rapid recovery.

The bulk of the work to be disposed of.

The problem of securing the safest and most efficient anaesthesia with the available anaesthetic agents and apparatus.

#### LACK OF PRELIMINARY PREPARATION

It is a more or less generally recognized fact that unless a patient is properly prepared for operation by preliminary catharsis and fasting the course of his anaesthesia will not run smoothly. We fail to appreciate this fact in our routine work and its significance is only apparent when an "immediate" operation is undertaken.

When practically all operations are immediates, as is the case in the Zone of the Advance and in many instances in the Intermediate Zone, we are constantly confronted by this situation. "Not only the bowels but the condition of the bladder requires attention. So many patients voided their urine while under nitrous oxide gas, a bottle was given them as a matter of routine immediately before" (Corfield, *Am. J. S.*, 1, 18).

As might be expected where nitrous oxide alone is the anaesthetic, vomiting is infrequent. "I never had any vomiting after this anaesthetic ( $N_2O$ ) and I dare say most of the patients had a stomach fairly full of food" (Corfield). On the other hand, the same author states, "It is a curious thing that in many patients who have had a meal just before they were wounded the process of digestion, or at any rate the stomach movement, is totally arrested and they will often vomit food ten or twelve hours after they have taken it. I remember one patient

who in the first stage of anæsthesia, started to vomit violently and brought up three bowlfuls of bully beef and biscuit. By the time he had finished he was fully conscious again, and I found out from him that he had had this meal twenty-eight hours before and that he had been wounded two hours after it."

A rather novel and interesting suggestion as to preliminary treatment may be noted in Corfield's communication: "I was always in favor of patients who could swallow at all, having a pint of hot tea while waiting operative treatment. It has the advantage of stimulating and warming them, they always like it and it is a very effective transfusion, too. Being liquid, even if vomited, no mechanical obstruction of the respiration is likely to follow." While the patient's *esprit de corps* doubtless improves with this treatment it is questionable whether any other benefit follows, for movements of the stomach usually cease during anæsthesia, and it is a physiological fact that water is not absorbed from the stomach, or, if absorbed, the quantity is so small as to be negligible (Howell, p. 719, 2d ed.).

The great majority of the patients one sees are unshaven. In this connection it might be well to emphasize the great advantage of placing a layer of cotton between the skin and the facepiece whenever a closed method is employed. This is particularly true of gas oxygen anæsthesia.

The blessé approaches his anæsthesia in a very different frame of mind from the ordinary civil patient. He has been for hours on intimate terms with death. He has seen his friend of yesterday spill his life's blood on the common dust or in his mind's eye he still beholds the savage bayonet poised above his breast. To such a one the tinkling of instruments implies ultimate recovery and the anæsthetic an immediate relief from urgent pain. Such a patient is prone to yield cheerfully to his anæsthetic and to suffer the minimum of excitement.

The routine use of morphine in the Dressing Stations and the Field Hospitals if occurring shortly before operation is a valuable aid towards securing a smooth induction. Soldiers exposed to all sorts of weather under the most trying conditions become victims of bronchial affections which render them particularly irritable to ether vapor. Continuous smoking increases this irritability, which preliminary morphine allays but does not always completely remove.

The injury which the patient has experienced, coupled with the subsequent exposure and loss of blood, tends to make the blessé an easy patient to anæsthetize.

The accidental and peculiar circumstances which preceded anæsthesia administered at the Field Hospitals and ambulances, as well as those at the Casualty Clearing Stations, are such as to counteract the need of the usual fast and catharsis, reducing or completely eliminating the period of excitement, shortening the period of rigidity and hastening the onset of complete relaxation.

Anæsthesia administered at the Base Hospitals and elsewhere in the Zone of the Interior closely resembles the anæsthesia ordinarily seen in civil life. Preliminary preparation may be painstaking and deliberate as most of the operations done here are planned in advance.

#### THE UNUSUAL AND URGENT NEED OF A SPEEDY INDUCTION AND RAPID RECOVERY

Where hundreds and thousands of wounded are to be briefly treated and passed on for further observations, it is essential that each patient be rendered unconscious and recover consciousness again as soon as possible. A slow induction takes time which should be used for operating, otherwise it takes longer to induce the anæsthesia than it does to do the operation. A slow recovery requires the presence of a nurse or an attendant who might be useful elsewhere, it also prevents a rapid evacuation to the Zone of the Interior.

In order to meet these conditions various agents and methods have been employed and found satisfactory. Ethyl chloride, nitrous oxide, nitrous oxide and oxygen, chloroform, chloroform ether mixtures and ether alone has been used for general anæsthesia. Novocaine, cocaine, stovaine for local and spinal methods.

For general anæsthesia gas oxygen is certainly the anæsthetic of choice. Its limited use has been due to the employment of complicated apparatus designed for constant flow methods instead of simple devices for use with an intermittent flow. Marshall (*Am. J. Surg.*, 1, 18) makes this clear when he says: "The ideal anæsthetic is one with which induction is rapid, and recovery complete in a few minutes after operation, so that the patient is in a fit condition for early evacuation by the ambulance train. Apparatus is subjected to much wear

and tear, so it should not be complicated or delicate. Of the anæsthetics I have used gas and oxygen meets these requirements best. Its only drawbacks are that the apparatus is somewhat cumbersome and the materials costly." Apparatus for the administration of gas oxygen anæsthesia by the method of intermittent flow is not cumbersome and the volume of gas consumed is so small that the method is not costly.

Ethyl chloride resembles gas oxygen in its speed of induction and its rapid recovery. It appears fairly safe in experienced hands. M. Boureau (*Presse Medical*, 21, Mai 17) recommends it for its non-toxic qualities, its rapid elimination and the ease with which it is accepted by the patient. It is especially useful as a first-aid anæsthetic; *i. e.*, ligation of blood-vessels, removal of large superficial foreign bodies, etc. Nothing could be more simple than the paraphernalia required, for a handkerchief is all that is necessary. The method employed is as follows: The patient is placed on the operating table and the preparation of the wound is begun. Two or three Cc. of ethyl chloride are sprayed on a handkerchief or upon a towel folded in four. This is placed loosely over the nose and mouth. The patient is instructed to breathe deeply. At the end of about half a minute the dose is renewed and the air supply is restricted. Induction is said to be complete in about two minutes. Maintenance may be carried on by the addition of one Cc. a minute. The signs of anæsthesia resemble those of chloroform. The respiration is tranquil and the pupils are moderately dilated, the corneal reflex is absent. Ethyl chloride may be said to be an anæsthetic of expediency rather than choice. Its action is more rapid than that of chloroform and attended with about an equal degree of risk.

*Chloroform ether mixtures*, so called mitigated ether, have been used with much success by McCardie, of London. Warm ether vapor given by the Shipway apparatus has also found favor with the British.

#### THE BULK OF THE WORK TO BE DISPOSED OF

Operative activity in the Zone of Advance and in the Intermediate Zone depends directly upon military activity in the particular zone under consideration. The surgery which is done in the Base Hospitals in the Zone of the Interior, drawing as it does its wounded from a wide territory, is of a more leisurely, more constant, and less acute nature.

Major Kellog Speed describes the methods in the average Casualty Clearing Station as follows: "In addition to administrative officers, etc., there are assigned to each Casualty Clearing Station surgical teams composed of picked men of surgical ability, each supplied with his own anæsthetist, operating nurse, and orderly. These teams divide the major and the minor surgical patients in rotation as fast as they are able to finish each operation. Team work is divided thus, each team works for eight hours a day except during rush times, when they are expected to do sixteen hours' duty. Three teams are on duty from 9 A.M. to 5 P.M. in the major theatre, using five or six operating tables; two teams work from 5 P.M. to 1 A.M., and one team from 1 A.M. to 9 A.M., under ordinary conditions. The work is continuous as long as the hospital is taking in. Teams cease to take on patients one-half hour before the expiration of their time so that their table may be cleaned up and prepared for the next group.

"The major operating theatre is centrally located. The minor theatre is near the dressing tent. All are electrically lighted. The arrangement of five or six tables in the major theatre permits the anæsthetist to move from patient to patient in advance of the operator while dressings and splints are being applied or the operation is being finished. The operating nurse assists the surgeon, the orderly brings in the supplies, helps with the dressings and cleans up the table. There is complete independence for each team."

The necessarily large amount of work to be done in a short time calls for skilful and speedy anæsthesia. Gwathmey writes as follows: "Nitrous oxide oxygen and ether is the only anæsthetic for this kind of work, as speed is the only desideratum. I have given as many as thirty-four anæsthetics in one day—something which would be impossible with chloroform or ether."

#### THE PROBLEM OF SECURING THE SAFEST AND MOST EFFICIENT ANÆSTHESIA WITH THE AVAILABLE ANÆSTHETIC AGENTS AND APPARATUS.

This problem is one of the great difficulties of military anæsthesia. Special supplies soon become exhausted and are difficult, if not impossible, to replace. Intricate apparatus often cannot be repaired on the spot and becomes worse than useless. The ability to improvise

apparatus is essential. Improvised apparatus can only be safely used by an experienced administrator, for experience must supply the elements of safety which the apparatus lacks. The Field Hospital, the Divisional Ambulance and the Casualty Clearing Station is no place to begin one's experience as an anæsthetist. The anæsthesia administered here must be expert from every point of view. Unless this be so an attempt to make speed will result in a stormy induction, an uneven maintenance under poor control and a delayed recovery. One must know by experience the limits of safety which the patient will tolerate. The experienced man will not be carried away by the allurements of a new agent or technic which is brought before his attention, as he will already have experienced fads and seen their early demise. The experienced man will be conservative, and for this reason safe. On the other hand, he will be so familiar with the various phenomena which normally appear with the unconsciousness of anæsthesia that the question of apparatus to bring about this state will be of secondary consideration. He will appreciate the value of preliminary medication and he will be in a position to diagnose preliminary medication which has not been reported and which influences the course of the anæsthesia. The military anæsthetist should not only know the best methods which are available for insufflation anæsthesia, but he should be able to improvise inhalation methods to take their place. He should be prepared to give good gas oxygen anæsthesia without the use of percentage devices dependent upon complicated reducing valves and heating attachments.

The difficulty, then, of accomplishing satisfactory anæsthesia with limited apparatus implies the need of a trained anæsthetist. As there is little likelihood of a sufficient number of trained anæsthetists being found in the enlisted personnel to supply the demands of the army and navy the Government is confronted with the problem of training anæsthetists for the various branches of its service.

Such training should be uniform. Guedel writes from the front as follows (*J. Surg.*, I, '18): "As for anæsthesia in the American army, this should be given some general attention, by all means, by the United States authorities. As it is, the methods are slipshod and careless to a degree that causes an enormous wastage of anæsthetic material and occasional accidents which are costly to the Government,

to say nothing of the occasional loss of life of an American soldier. As it is, the surgeon, no matter what his experience or rank, has full control of the anaesthesia for his cases, and as a rule he knows nothing of anaesthesia. With such surgeons the co-operation of skilled anaesthetists is a secondary consideration. This matter should be, and could be, regulated to the great advantage of all concerned. A system of suggestions and instructions to the medical corps of the army in general, based upon observations of the needs of this particular war in the matter of surgical anaesthesia, would go far towards saving money, time and life. . . . Every scientific branch of the service in the army at this time is advancing except anaesthesia, and that is still regulated by rule of thumb."

In anticipation of this situation the author made the following suggestions October 15, 1917:

That a school of anaesthesia be established in New York City.

That this school be under the control of a dean, who would assume responsibility for the theoretical preparation of the students and who would give actual demonstrations whenever possible. A standard textbook to be employed and the most efficient methods as well as methods of expediency to be taught.

That upon the completion of the theoretical training, which would not exceed four or five days, the student be detailed to a large city hospital as a regular interne anaesthetist for a period of three months. These students would, by continual replacements, fill in the gaps left by internes who have joined the medical reserve. They would serve the hospital and the army simultaneously.

That each interne anaesthetist be accompanied by an assistant who would, by observation, prepare herself (woman physician) for actual administrations.

That these interne anaesthetists be under the immediate supervision of the dean and competent assistants appointed by him.

On the proposed basis in New York City between forty and fifty competent anaesthetists could be turned out each month, or between five hundred and six hundred in the course of a year.

Beginning in the fall of 1917, a course of lectures and demonstrations has been in progress at the Rockefeller War Demonstration Hospital. An attempt has been made to explain and demonstrate insuf-

flation and inhalation anaesthesia to the various groups of army officers who come to this institution for instruction.

Briefly, then, in military practice, *many unprepared patients must be speedily anaesthetized, frequently with improvised apparatus in such a manner that their recovery is complete and rapid.*

**OBSERVATIONS ON THE TECHNIC EMPLOYED IN THE ADMINISTRATION  
OF ETHER, CHLOROFORM, NITROUS OXIDE, NITROUS OXIDE AND  
OXYGEN, ETHYL CHLORIDE SPINAL ANÆSTHESIA AND HYPNOTISM  
ON THE WESTERN FRONT**

Ether is universally used, it is employed as the terminal anaesthetic in about 80 per cent. of all cases. It is argued that the ether manufactured in Europe is irritating, non-volatile and inefficient. These characteristics are emphasized when used with an open mask. "It is not much better than our wash ether at home," writes Guedel. "It is difficult, indeed, to put a patient to sleep with it, to say nothing of securing a quiet state of anaesthesia. From the coughing and the great quantities of mucous secreted, it would seem to contain more sulphuric acid and formalin than anything else. Also it is about as volatile as alcohol. You never get any frosting on the mask. Usually a patient will walk right out from under anaesthesia with this ether in spite of continuous administration and a clean mask becomes soggy and useless after about ten minutes. Whether or not this ether is the usual European ether I am not certain, but from many inquiries that I have made I am inclined to believe that is is."

When a closed method is used, however, these difficulties become less apparent. The apparatus of Obredanne is popular with the French. It resembles the Clover and the Bennett in its general characteristics.

The Shipway apparatus is quite popular with the British. It has proven especially useful in head and neck work.

Chloroform ether anaesthesia has been extensively employed by McCardie, of London. After numerous experiments it was found that the proportion  $E_{16}C_1$  gave the greatest satisfaction. This mixture was used in about 1200 cases. McCardie applies the term "mitigated ether" to this mixture. It is used as one would straight ether, in a closed apparatus. McCardie's conclusions are as follows:

The irritation caused by ether vapor administered by the closed method is much mitigated by the addition of very small amounts of chloroform.

The addition of this small amount of chloroform to ether distinctly saves the work of the lungs and heart, which during a long or severe operation may be a very important factor.

The mixture is practically as safe as the administration of ether alone, because ether greatly predominates.

It is valuable as a routine method of producing anesthesia in soldiers, being reasonably safe and rapid.

A few drops of  $E_2C_1$  are preferable to ethyl chloride as a preliminary to the administration of ether or  $E_{16}C_1$ .

For maintenance of anesthesia  $E_2C_1$  or chloroform are preferable to even mitigated ether, owing to the prevalence of respiratory irritability among soldiers.

#### CHLOROFORM

Bilhaut, of the Hôpital International de Paris, finds chloroform easier to administer than ether and less likely than ether to cause chilling of the respiratory tract such as may produce pneumonia or pulmonary congestions. In 812 important operations performed by Bilhaut there were no unhappy results from the use of chloroform. The reaction on the liver has been found negligible. Fissinger has called attention to the fact that the results obtained by Bilhaut seem to prove that the operations performed on soldiers under war conditions give better results than in times of peace. It seems as if the new conditions created by the war have brought about an increase in the potency of organic defense. Under ordinary conditions it seems that ether might possibly be superior as an anesthetic to chloroform, since it does not cause alterations of the hepatic cells; however, it would appear, from the communications of Bilhaut, that under the existing conditions, chloroform has lost part of its injurious properties.

This is interesting, in view of the extreme anti-chloroform attitude assumed by the profession at large.

Guedel states: "I have come to the point that with all my anti-chloroform prejudices I am using chloroform in all cases when I cannot get American ether. They (the French) use it about as we do

ether back home. They don't seem to fear it at all, but that may be because of the extremely low price placed on human life in this war. They have their accidents, of course, but seem to think them infrequent."

Corfield has used chloroform for inducing anæsthesia as follows: An open drop mask is covered with an inside layer of lint, followed by five or six layers of gauze. A dram of chloroform is poured on the inside of the mask. While the patient's respiration is being accelerated by conversation the mask is gradually lowered to the face, ether is then slowly dropped upon it and soon increased to the limit of toleration. "By this method," Corfield states, "I rarely got the slightest movement on the part of the patient and he was generally ready for operation in two minutes from the beginning. . . . During my six months I never had a fatality or, indeed, any case in which the method gave me anxiety. The advantages of hyocine and morphine for this purpose (preliminary medication) are common knowledge, and I need not dilate upon the method beyond saying that it was particularly useful with us, for it kept patients quiet two or three hours after they got back to the ward and so saved the attention of a nurse or orderly."

#### NITROUS OXIDE, NITROUS OXIDE OXYGEN

"From my point of view," writes Corfield, "patients were divided into two classes, those necessitating a short anæsthesia, up to ten or twelve minutes, who were given nitrous oxide, and those requiring a longer period, who were given chloroform and ether. The first class comprised wounds to be cut out and dressed, foreign bodies removed and guillotine amputations, and for such cases nitrous oxide was used. The advantages were both a saving of time and of labor. Time was saved because the period of induction and returning consciousness were a matter of seconds rather than minutes and labor was saved because most of these patients could walk back to their own wards, either by themselves or by the help of one orderly, whereas chloroform or ether would have meant that every case would be a stretcher one. For prolonging nitrous anæsthesia one had to use a gas and air mixture. My method was to get them deeply under and then push back the air valve for a quarter to a third of an inch so that the patient

would get sufficient oxygen to keep him from asphyxiation. Patients varied in the amounts they required. It was, one might say, a compromise between color and consciousness; with too much air they became sensitive to pain, and with too little air they became cyanotic. Occasionally one came across a patient who was difficult to keep under, because of clonic spasm and rigidity, and in my experience nitrous oxide is a difficult anaesthetic to give unless a patient is lying on his back."

The importance of substituting oxygen for the air which is employed in nitrous oxide air anaesthesia is rapidly being appreciated by European anaesthetists. The resulting gas oxygen anaesthesia is gradually finding its proper place as an invaluable anaesthetic agent in military surgery. The delay in this movement may be explained by the fact that the majority of those who gave anaesthetics at the beginning of the war were not all-round experienced anaesthetists. Had they been so the great value of gas oxygen anaesthesia in selected cases would have been well known to them and would have been employed at the start.

The English were the first to introduce the use of gas oxygen in the military surgery of this war. The simplest methods were used with success. The Clover inhaler and other closed devices were employed, the gases being fed intermittently and rebreathing being practiced. The advent of America was followed by a marked impetus in gas oxygen anaesthesia. Two problems confronted the department of surgical supplies. The first was—what type of machine shall we provide?—secondly, how shall we provide the necessary gas for these machines? Steps have been taken to establish a gas oxygen plant in France, where the nitrous oxide and oxygen may be manufactured on the spot. Guedel, writing from the Mackey-Roosevelt Base Hospital, in France, states that: "Nitrous oxide here is almost out of the question. I understand that Major Crile brought a lot of it over for the Lakeside Unit, but I do not know of any available source here at our part of the front . . . A number of Units have gas apparatus but at present nothing to use in them."

It is quite likely that since this communication was sent arrangements have been perfected for an adequate supply of both gases.

Most of the apparatus sent from America to France and England

for the administration of gas and oxygen is complicated in structure, designed for constant flow methods and intended to be as nearly as possible automatic in action. These machines are for the most part accurately and painstakingly made. It has been attempted in the construction of these machines to do away, if possible, with the need of an experienced physician anæsthetist. In view of the shortage of anæsthetists, such an attempt is certainly justifiable if the perfect delivery of gases was the solution of the problem. A number of first-class machines are in use, among which may be mentioned the Gwathmey, Connell, McKesson, Ohio and Heidbrink.

The desirability of gas oxygen anæsthesia has been repeatedly pointed out in foregoing paragraphs, especially as applied to the work of the Field Hospital and the Evacuation Hospital. The simplicity and economy of method demanded by the situation does not appear to have been properly met, however, as might well be done if intermittent flow methods with rebreathing were adhered to.

#### ETHYL CHLORIDE

M. Boureau recommends ethyl chloride for the Divisional Ambulances and Field Hospitals as well as for operations in the Base Hospitals. Induction is speedy and recovery rapid and complete. The patients are shocked, more or less tired mentally and physically from continual tension. They often suffer from hemorrhage and are generally depressed. The most courageous does not wish to suffer further pain. One should not hesitate, therefore, to put him to sleep whenever necessary. Ethyl chloride is recommended as the anæsthetic of choice. It is well borne and chosen by the patient, where ether or chloroform have been previously used. Boureau has made use of ethyl chloride as many as fifteen times on the same case for the application of painful dressings. The recovery from the anæsthetic is proportional to the size of the dose. If administered less than ten minutes the recovery is rapid. If more than ten minutes the period of recovery may extend to ten or fifteen minutes. There may or may not be slight vomiting after the recovery. It appears to be especially useful in pulmonary cases.

Savariaud employs ethyl chloride for the dressing of large painful shell wounds. He makes use of the following simple technic:

The patient's eyes are protected by a pad of four or five thicknesses of gauze. A large square of oiled silk is then placed over the head of sufficient size to cover the face and allow the four corners to be tucked under the head. The tip of the ethyl chloride tube is then inserted through a small hole in the silk and the drug is slowly sprayed within. Anæsthesia may be maintained with about one Cc. of ethyl chloride a minute.

#### SPINAL ANÆSTHESIA

B. Desplas, in an article entitled, "Anæsthesia a la Stovaine en Chirurgie de Guerre," extols spinal anæsthesia as easy to administer, rapid, economical, may be repeated, good in emergency work, and does not demand the presence of a specialist.

He employs stovaine in doses of 5 cgm. (ampoules de Billon), injected between the fourth and fifth lumbar vertebrae.

Instead of entertaining his patient by conversation or by allowing him to smoke or view the operation, he recommends absolute silence, has the patient blindfolded, and cotton placed in his ears. If slight pallor or nausea appears the patient is instructed to breathe deeply and is given something warm to drink.

With 297 anæsthesias he has had no deaths or unpleasant experiences. Death, when occurring, is ascribed to three causes: Incorrect injection, improper solution of unknown strength, and the use of cocaine.

The stovaine is eliminated by the urine in from six to nine hours. It has no effect on the urinary secretion.

G. Marshall, writing from a Casualty Clearing Station, states: "We require a method which will not be harmful to a patient suffering from the shock of injury, and one which will minimize the shock of operation. It has been urged that spinal anæsthesia would meet these requirements, and would, therefore, be of great value in military surgery. For men wounded in the lower extremities I found it a convenient and satisfactory method at a Base Hospital; cases of profound collapse did not occur. The same good results were obtained at a Clearing Station in all patients who had been wounded not less than forty hours before operation. Of the more recently wounded, however, more than half showed signs of cerebral anæmia, with great fall of blood pressure shortly after intrathecal injection of stovaine. These

signs were pallor, nausea, retching, vomiting and loss of consciousness. More rarely I have seen extreme restlessness and in one case convulsions. The radial pulse disappears and the patients present an alarming picture of collapse, which may necessitate interruption of the operation. It has been stated that collapse under spinal anaesthesia is not dangerous. I have seen two cases in which it proved fatal, and have heard of a number of similar fatalities in recently wounded men. The appearance of the patient is of little assistance, the pulse rate and the blood pressure do not help us at all. A valuable indication is obtained by determining the concentration of the blood. I employ a Haldane Hemoglobinometer. In practice I find that if a recently wounded man has a hemoglobin percentage of over a hundred it is safe to administer stovaine intrathecally. If the reading is below a hundred he will certainly show a serious fall of blood pressure and symptoms of collapse. Stovaine should not be administered intrathecally to men who have been wounded less than forty hours, unless it has been demonstrated that their blood is of normal concentration."

Intravenous ether anaesthesia, regional intravenous anaesthesia, local anaesthesia and hypnotism have all been used in the military surgery of the present war. In war conditions Podiapolksky has found that the men responded with exceptional facility to *hypnosis*; he found only about two per cent. quite refractory. He has found it useful in the wounded of all the nationalities that he has encountered. This treatment is rather for the sensory crisis of psychic origin than for anaesthesia for major operations.

The most striking fact brought out by the increasing number of articles dealing with "the anaesthesia at the front" is the complete diversity of views as to the best anaesthetic agent and technic of administration to be used as a routine under given conditions. Straight ether by the open and semi-open method, chloroform, ether mixtures, ethyl chloride, nitrous oxide oxygen, spinal anaesthesia, and even hypnotism, as we have seen, each has its earnest advocate, who uses his method largely to the exclusion of others.

One might conclude from this condition of affairs that one agent and method is about as good as another, that the apparent success of all methods is due to a keen specialization of the particular method

in question, or that the agent and the method employed is merely the result of accident, nothing else having been available.

The first consideration, that one anaesthetic agent and method is as good as another will find credence only with those who are unacquainted with the subject. For the ability to choose the anaesthetic and its method of administration is the mark which distinguishes the physician anaesthetist from the mere lay technician or so-called nurse anaesthetist.

The second consideration, that the apparent success of all methods is due to specialization of the method in question, is certainly true to a considerable degree. Practice makes perfect here as in any other division of labor. By constant application anyone of ordinary manual dexterity can give an entirely satisfactory anaesthesia with ether, chloroform, ethyl chloride, or even with gas and oxygen. If this were the end of the matter no harm would be done; the difficulty lies in these administrators heralding their work as a discovery and inviting the uninitiated to follow in their steps. The worth of any anaesthetic agent can only be determined by the aggregate experience of many workers. It is only in this fashion that we may anticipate and guard against fatalities in the work of the average anaesthetist. It is, therefore, not fair to conclude that specialization in the use of a single agent will control the safety of that particular drug, that experience alone is all that is essential to make a drug safe.

The third consideration, that the agent and the method employed are merely the result of accident, nothing else having been available, may be true where there is a constantly shifting personnel, as is likely to occur in the Field Hospital and the Casualty Clearing Station. In the Base Hospitals, on the contrary, the personnel is more or less fixed and the selection of the anaesthetic may be painstaking and deliberate. The literature which has appeared has been of such a character as to emphasize the fact that the worker at the Base Hospital has had not only the time but the inclination to work out the problems of anaesthesia in his particular case. In many instances, however, the choice of the anaesthetic agent and the method of administration is suggestive of the fact that the administrator was not trained in the fundamentals of anaesthesia before he began to administer anaesthetics for military purposes.

The diversity of opinion, then, which shows itself in the literature is quite natural and interesting; but—is not the individual worker myopic, so to speak, because of his concentration and the demands placed upon him? It would seem safer to judge the entire field at a distance and to weigh the various agents and methods in the balance of universal experience.

Surgical methods and technic changes with war conditions.

We find antisepsis in wounds superseding asepsis. Open framework "airplane splints" are employed in place of the familiar plaster of Paris. In medicine, shell shock opens up a large field for the psychiatrist. Anæsthesia, however, when accommodated to the difficulties mentioned in the first part of this chapter, namely, the need of a rapid induction and recovery in a large number of poorly-prepared patients, often with improvised apparatus, remains essentially the same as it was before the war. The underlying principles for inducing and maintaining anæsthesia are the same, and the accepted methods of securing rapid and complete recovery are identical. The need of anæsthesia in wounded cases, as pointed out by M. H. Vignes (*Presse Medical*, Dec 4, '16), is even greater than in times of peace. "It is absolutely necessary to protect patients from pain. Anæsthesia alone places the patient in the best condition and allows the performance of good surgery, and, of even greater importance, it prevents shock incidental to surgical manipulation, which has been added to that of trauma. One can die of pain. It is essential to be as economical in the loss of nervous energy as in the loss of blood. The ambulances should, therefore, have trained anæsthetists, and perfected appliances should be carried to the front."

In the light of the facts which have been considered, it would seem just to assume the following conclusions:

That a department of anæsthesia be established by the Government which shall include in its composition experienced anæsthetists.

That the department standardize, on a basis of universal experience, the safest and most efficient anæsthetic agents, methods and technic to be employed under given conditions.

That practical and theoretical instruction in anæsthesia be disseminated by this board through sources available for its use; *i. e.*,

experienced men at work in large cities to teach in hospitals of 100 beds or more, students detailed for this work by the department.

That this instruction be arranged with a view of meeting the difficulties outlined in the first part of this chapter.

That the necessary apparatus be purchased upon its intrinsic value as indicated by the peculiar conditions to be met, and that the practice of wholesale purchase of apparatus unfamiliar to the prospective administrators be utterly deprecated.

In view of the experience of those at the front it would also seem fair to assume:

That in the Zone of Advance and in the Intermediate Zone the anaesthetic indicated for incomplete anaesthesia is gas and oxygen, administered in the simplest manner, namely, by the method of intermittent flow with rebreathing. Complete anaesthesia to be brought about and maintained by ether given with a closed method, recovery to be ushered in and completed by a return to gas and oxygen.

When gas and oxygen is not available a chloroform, ether, ethyl chloride or chloroform anaesthesia, may be employed for incomplete anaesthesia, which may be made complete and maintained by ether given by a semi-open or closed method.

That spinal, intravenous, regional or morphine hyoscine anaesthesia, *per se*, be reserved as methods of expediency to be used by an experienced administrator.

That in the Zone of Advance and in the Intermediate Zone inhalation methods be the methods of choice. Intrapharyngeal and intratracheal inhalation anaesthesia for head and neck work by the tin-can method, to be used in preference to complicated insufflation methods.

On the other hand, in the Zone of the Interior, the Base Hospitals should have the best and most complete equipment. Gas oxygen by constant-flow methods may here be used if desired. Insufflation anaesthesia by the Connell Anæsthetometer should be the method of choice. At the Base Hospital arrangements should be made to instruct as well as to anaesthetize. The most complete ante- and post-operative treatment may here be carried out, with every assurance of success.

## FURTHER STUDY OF THE EFFECTS OF PAIN ON THE CELLS OF THE CENTRAL NERVOUS SYSTEM

By G. S. FOSTER, M.D.

Surgeon and Pathologist to the Hospital Notre Dame de Lourdes,  
Manchester, New Hampshire

### PART I

FOR the past four years we have, in our clinic, attempted to observe closely the effects of pain upon the central nervous systems of surgical cases. During this period the average mortality in these cases has been one and a half per cent. In most of the cases which have died we have been unable to obtain an autopsy. Thus the direct study of the human nervous tissue, in so far as microscopical pathology is concerned, has not been very fruitful. On the other hand, if we had been able to obtain a post mortem examination in every fatal case, the statistics gathered would have been far from a true guide to the situation, because the percentage was too low to be of any benefit in controlling those who recovered. In itself this is rather a gratifying circumstance.

Nerve block is the essential factor in the modus operandi. Just how this is accomplished does not matter so long as we can completely curtail any nerve transmission. Therefore, since we wished to give our patients every known preventive or prophylaxis against any surgical shock or reaction, we have used guinea pigs for the purposes of study and control. Furthermore, in this study, we have laid special stress upon the variations caused by the use of some narcotic, such as morphine with atropine or scopolamine, as the case might warrant; by the use of a general anaesthetic without the narcotic, and by the production of pain when neither narcotic or anaesthetic was used. This brief report is made in order to sum up these three conditions.

I personally have used Crile's method of analgesia for several years, and the habit has grown so fixed that when I witness operations without its use I can almost feel the pain of the incision.

As shown in our previous publications, cell nephelation seems a

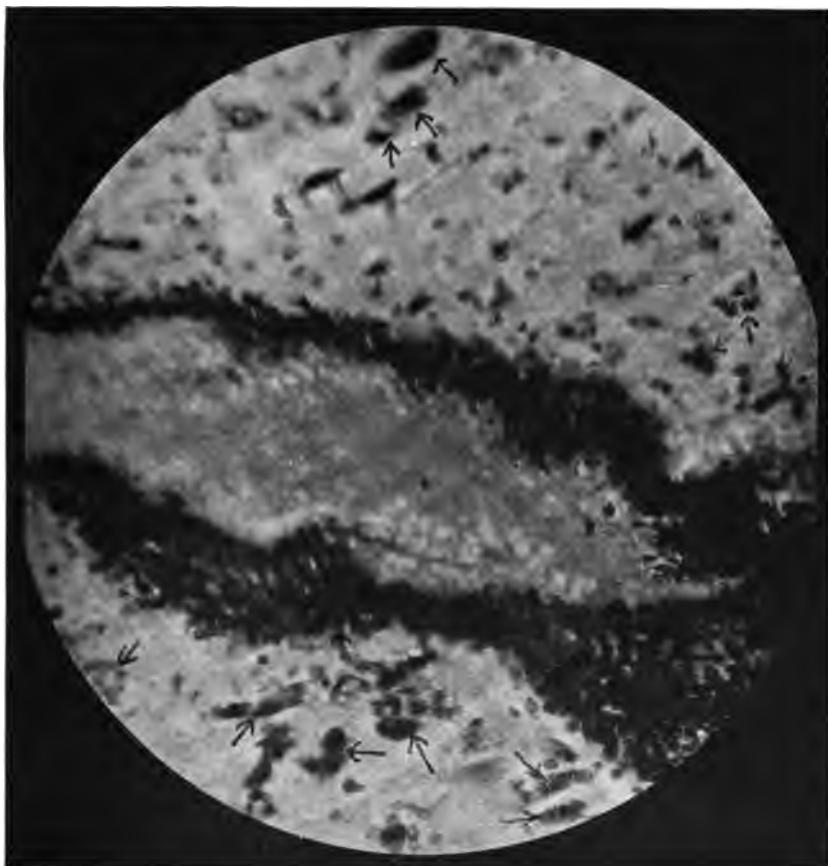
direct result of pain transmission along some nerve root or roots. If we can prevent this nephelation, we believe that we have prevented nerve transmission. Our previous study warrants us in thinking that nephelation means part or entire cell destruction. Some cells which have undergone only slight nephelation may completely recover after a time, but even this slight amount of injury will not be healed very quickly.

How many surgical cases become nervous and unset for a time following the operation! Every surgeon has seen this neurotic element in a certain number of cases, but fortunately the majority of cases have the normal resistance or supernumerary higher nerve cells, to overcome any neurosis. A certain number of surgical cases develop the nerves and take a long time to recover from them, and if we can in any way prophylact these cases by analgesia and amnesia we have accomplished something worth while. It is for this purpose and in behalf of these cases that we have studied these three conditions:

1. *Guinea Pig Operated Upon Under Ether Anæsthesia Only.*—No narcotic was given or analgesia used. This operation lasted thirty minutes. The usual abdominal incision was made and no attention given to the prevention of pain transmission. Figs. 1, 2, and 3 will show the results obtained from sections of the cord. Note the great number of nephelated cells. Some of these cells have even reached the stage of vacuole formation. These illustrations clearly define the pain transmission which occurs when ether anæsthesia alone is used, because a general anæsthetic does nothing but produce artificial sleep accompanied by the loss of reflexes. The pain transmission is present just the same, as is clearly shown by the photomicrographs. If it were not for the fright and fear on the part of the patient and the fact that they would be compelled to use their reflexes for the purpose of protection, we could operate without any anæsthetic and do no more harm to the nervous system. Any general anæsthetic merely eliminates the fright and obliterates the reflexes. The pain is there just the same and we get the resulting pain transmission along the nerve trunk.

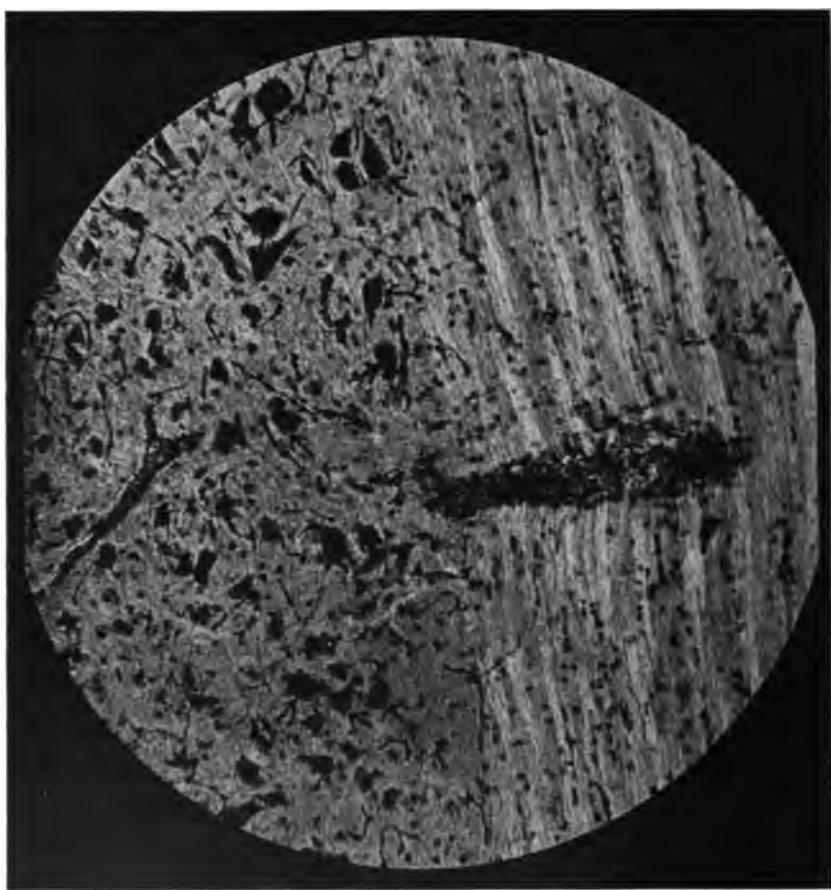
2. *Guinea Pig Operated Upon With the Use of Narcotics Only.*—Morphine and scopolamine were used, thus producing narcosis and amnesia. Figs. 4, 5, and 6 will show the results upon the nerve cells

FIG. 1.



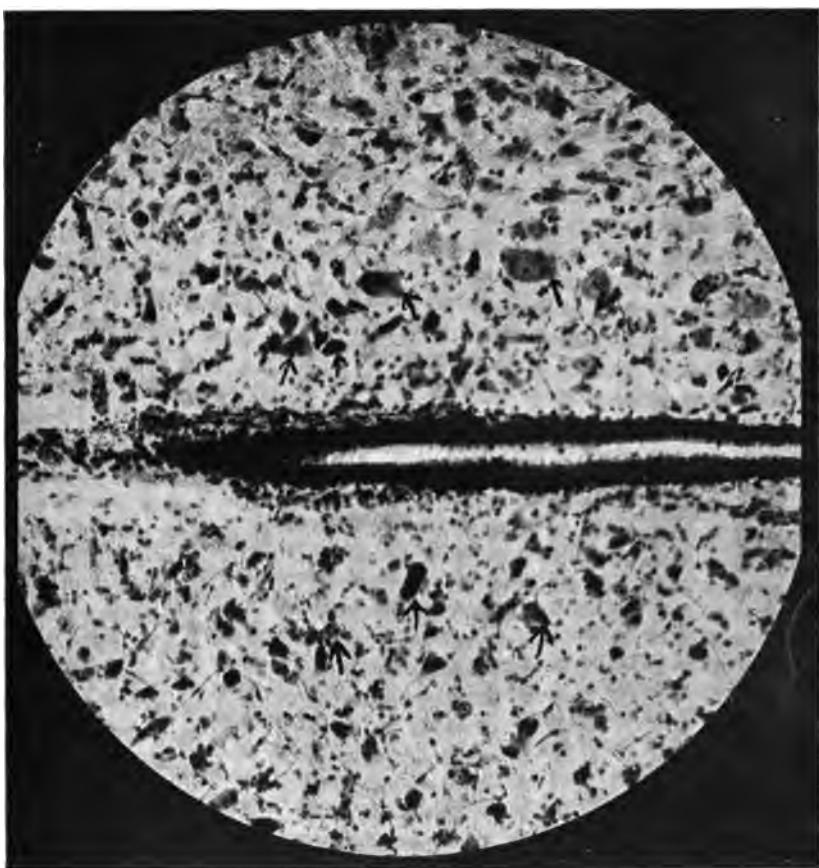
Guinea pig operated upon under ether anesthesia only. Section of cord. Note: Great number of nephelated cells. Some have gone to vacuolation stage. Nerve pain transmission extreme.

FIG. 2.



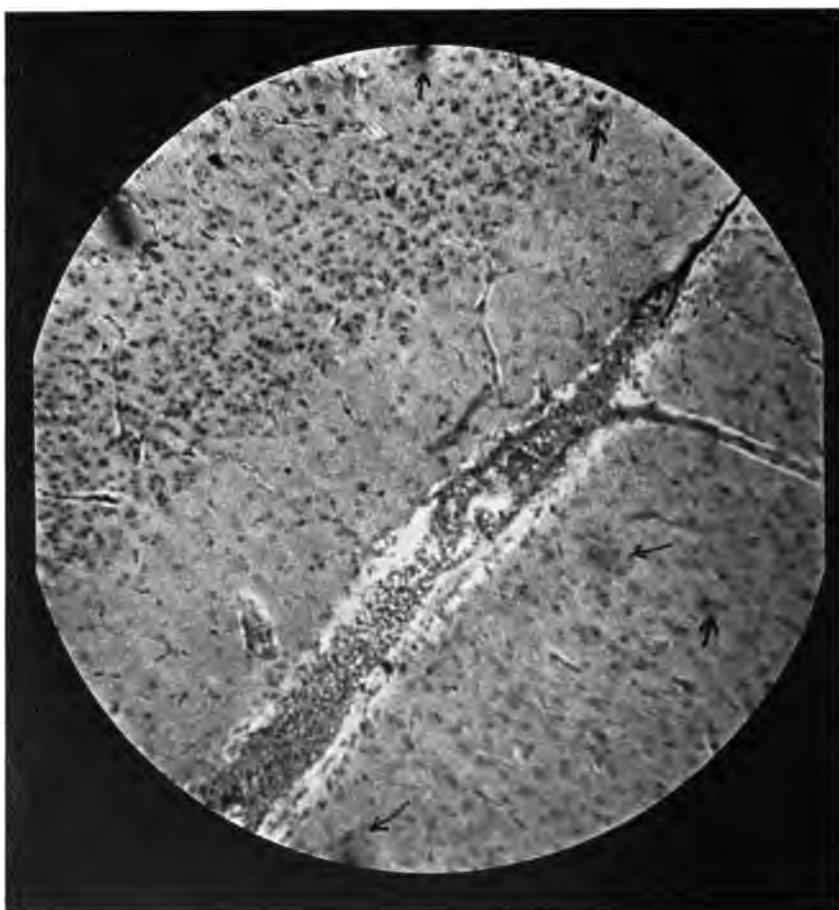
Guinea pig operated upon under ether anesthesia only. Section of cord. Note: Clearly defined nephelation and vacuolation. Nerve pain transmission extreme.

FIG. 3.



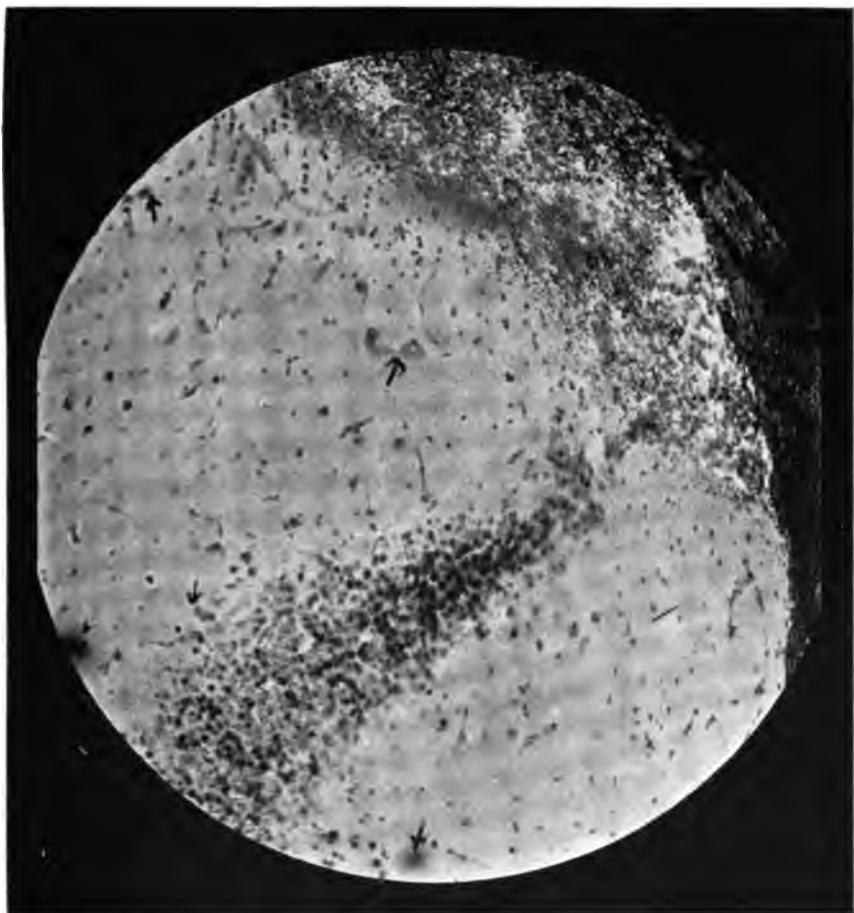
Guinea-pig operated upon under ether anesthesia only. Section of cord. Note: Great number of well-defined nephelated cells. Nerve pain transmission extreme.

FIG. 4.



Guinea pig operated upon under complete narcosis. No general anæsthetic or local analgesia. Section of cerebral cortex. Note: Nephelated cells indicated by arrow heads. No vacuolation. Many clearly-defined normal cells present in contrast to previous illustrations.

FIG. 5.



Guinea pig operated upon under complete narcosis and amnesia. No general anesthetic or local analgesia. Section of cerebral cortex. Note: Nephelated cells very discrete. No vacuolation. Cellular nephelation shows less degree of involvement. Compare 1, 2, 3. Many normal cells present.

FIG. 6.

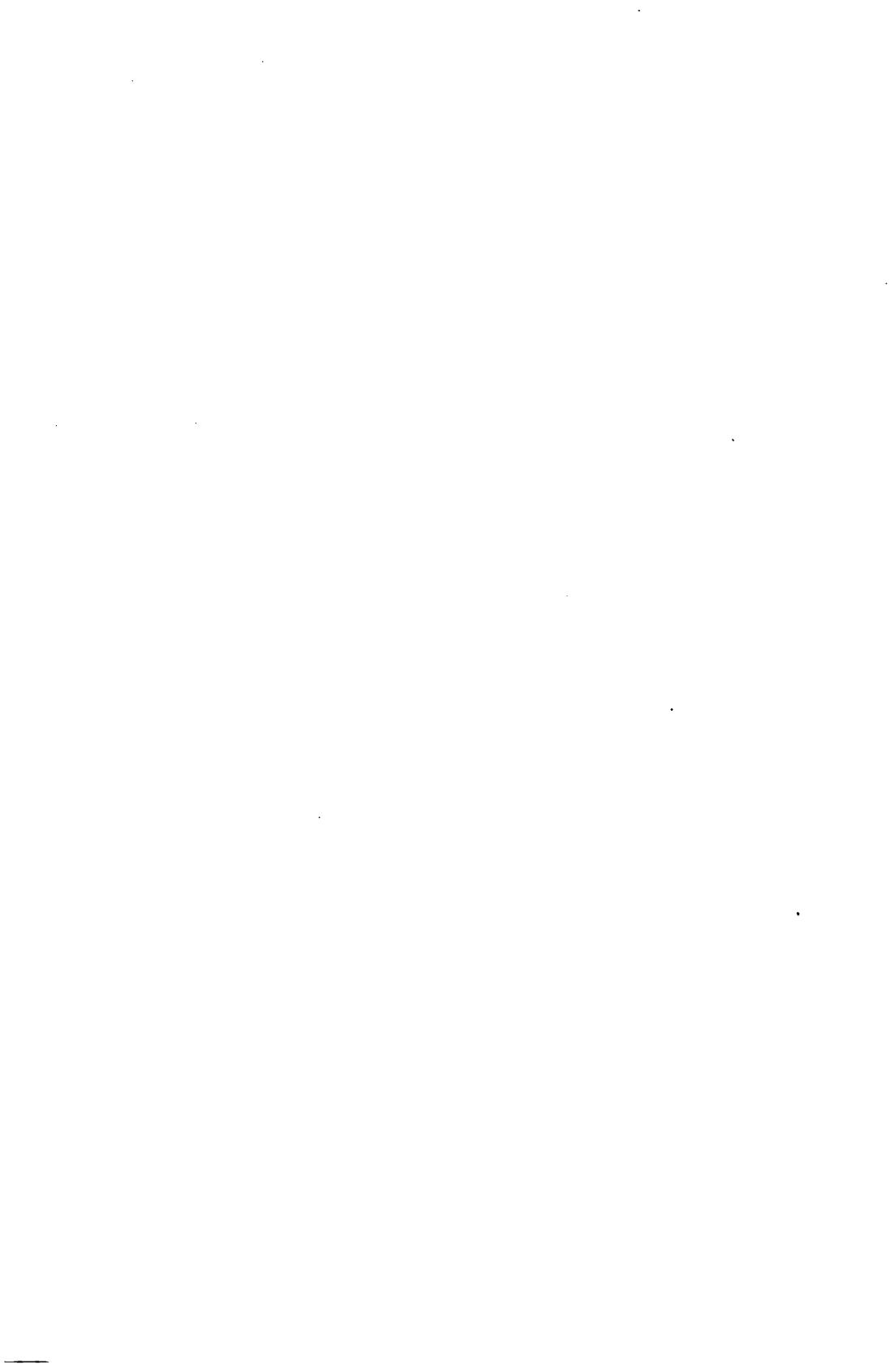


Guinea pig operated upon under complete narcoeis and amnesia. No general anæsthetic or local analgesia. Section of cerebral cortex. Note: Discrete nephelation. No vacuolation. Many normal cells. Pain nerve transmission much less marked as compared to 1, 2, 3.

FIG. 7.



Guinea pig operated upon under local analgesia alone. No narcosis was produced, no general anesthetic given. Section of cord. Note: Only an occasional nephelated cell. The field is filled with normal, unhampered cells. What nephelation is present could be placed at the door of fright. Compare with previous illustrations.



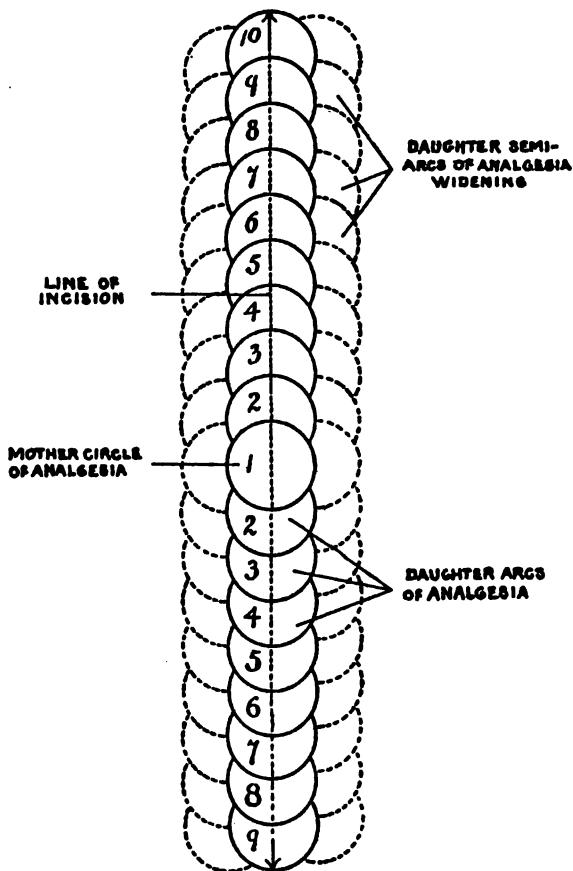
of the cortex of the brain. Nephelation is present to a much less degree than under the general anæsthetic and no vacuole formation shows in this condition. By this, we can see that narcosis and amnesia retard pain transmission along nerve trunks and the effect upon the nerve cells much more than does the general anæsthetic. Evidently the shock to the nervous system is much less when we produce narcosis and amnesia without any general anæsthetic. In the latter use fright or fear only are eliminated, accompanied by the loss of protection of reflexes. Just how much of the nephelation in condition two is due to fright or fear is hard to estimate. Possibly much of it is due to this and but very little to real pain transmission. If this be the case, the narcosis and amnesia are both supreme adjuncts in any operative procedure. At least our observations during these studies would warrant us in believing this to be true.

3. *Guinea Pig Operated Upon With Analgesia Alone.*—No general anæsthetic, narcosis or amnesia was used or produced but great pains were taken to block every avenue for nerve transmission. This blocking was accomplished progressively in all directions, and accomplished slowly by circular and daughter circular areas springing from the periphery of the mother circular area. This special formation is one of the staunch points which should be adhered to in the technic. Previously to the beginning of this operation each nerve distribution was tested out and the limitations of its function determined (five-tenths of one per cent. novocaine solution was used to produce the analgesia). Figs. 7, 8, 9 and 10.

This condition was produced slowly and under very noiseless conditions. This is another most important factor in the technic of production. I have learned that complete analgesia may be produced, yet if there is considerable noise the subject is kept nervously excited, and the success is not complete. Analgesia eliminates pain, yet the sense of pressure is present. Pressure, torsion and tugging are one and the same thing when working in this field. A same sensation is produced, the sense of pressure or touch. Patients will say at times that they feel no real pain but the pressure or tugging produced a very uncomfortable condition and at times gives the sense of nausea. With the production of this sense of nausea, the patient loses the fortitude needed for self control. Every layer of tissue should be handled

carefully and no undue pressure, torsion or tugging produced. Sponging of tissue, the use of retractors, clamping of arteries and pulling upon such structures as mesentery, omentum or the pedicle of growths should be guarded against.

FIG. 10.



Showing production of mother circle of analgesia with progressively formed daughter arcs for lengthening area and semi-daughter arcs for widening area. Each arc or semi-arc is made by the needle passing through the periphery of the previous arc or semi-arc.

The results of the observations in this condition show how careful we must be when operating upon a patient under a general anaesthetic only. That the patient is unconscious is no reason that we should be slow or use unwarranted roughness in the handling of the structures we may be working upon. The more careful we are with

all structures under any circumstances, the better results we will get for the patient in post-operative comfort and loss of shock.

#### CONCLUSIONS

By carefully studying the various illustrations of each condition produced the following conclusions are reached:

1. *General Anesthetic Only.*—Nephelation of the cells of the brain cortex and cord is marked.

Extreme degree of influence is shown by the unusual state of vacuole formation produced.

Any incision without amnesia, narcosis or analgesia, any one or all, produces a high degree of nerve pain transmission.

This production of nerve pain transmission produces shock, complete or partial, as shown by the cellular involvement and destruction.

Recovery from nephelation takes from four to ten days, time most important in the recovery of the patient. Cortex function cannot be normal again until this nephelation is absorbed.

Vacuole formation is permanent, as we have shown in previous articles, and the cells so involved never recover their function.

2. *Narcosis and Amnesia Only.*—Nephelation is quite marked but not carried to the degree produced in condition one.

The tendency is toward minimum nephelation, slight cell agglutination and no coalescence or vacuole formation.

The degree of harm produced or shock present is less than that in condition one.

By previous studies, we have shown that recovery from this degree of injury takes from one to three days. The cells return to normal appearance much earlier.

Narcosis and amnesia prophylactates shock and cortex injury to a higher degree than does a general anesthetic alone.

If we consider the patient, narcosis and amnesia are to be preferred to a general anesthetic if only one can be used, for the influence for good is greater with the use of the former than the latter.

3. *Analgesia Alone.*—Careful study of the illustrations will show that with complete blocking of nerve pain transmission no nephelation is produced in the cells of the cord or cerebral cortex.

This shows that analgesia is essential if we are to prevent pain nerve transmission.

Pain nerve transmission is the same whether the patient is conscious or not, unless nerve block is complete.

Interpretation of pain nerve transmission with its resulting pathology, nephelation, is always present without this condition.

From the patient's viewpoint, analgesia gives the best results with conditions two and one coming next.

No shock or cortex involvement is present in this condition.

*Résumé.*—Any general anæsthetic alone is no guard against pain nerve transmission. It merely produces unconsciousness and freedom from apprehension and worry.

Narcosis and amnesia are better conservers of nerve cell involvement than a general anæsthetic alone.

General anæsthetic, narcosis and amnesia give fairly good results in so far as pain nerve transmission and cerebral cortex cell involvement are concerned.

Analgesia alone under proper technic will prevent pain nerve transmission.

General anæsthesia, narcosis, amnesia and analgesia combined produce a perfect condition for the patient.

By it the conservation of the cord and the cerebral cortex involvement is reached.

Care in handling the patient and the parts involved cause the best results.

It is our humane duty to always consider the comfort of the patient.

## PART II

### THE USEFULNESS OF ANÆSTHETICS, LOCAL, GENERAL AND SPINAL, TO CONTROL PAIN TRANSMISSION IN THE NERVES

Previous papers by the author on the subject of nerve pain transmission have dealt with the subject from a general point of view. The purpose of this paper is to draw conclusions from experiments on guinea pigs under the influence of various anæsthetics as to the amount of nerve pain transmission in a special sense.

These experiments cover a period of careful study for the last

four years and a careful record as to the results obtained kept. All the pigs were kept under the most healthful and hygienic conditions. Only the best stock of hardy animals was used. Abhorrence on the part of a guinea pig to any one or more of the anæsthetics has not been taken into consideration. The weight of each animal was carefully kept and only full-grown animals weighing from thirty to sixty grams of each other have been employed.

Every precaution has been taken to carry out painstaking technic in each instance, and carelessness has not been tolerated. These little animals have been handled as carefully as if they were human beings and every kindness has been shown them. Therefore the conclusions have been drawn from as faithful work as could have been done by any conscientious and truly interested worker.

The purpose of these experiments was to show the true influence upon cord and cerebral cortex cells, upon striated muscle and liver cells when similar operations were performed under the influence of the various anæsthetics.

All nephelation is the control of the degree of pain nerve transmission shown by these experiments comparing all the degrees of change with the normal cell of the same form of organismal structure. The previous papers dealing with nerve block and pain nerve transmission with the resulting nephelation have shown that the degree of individual cell influence is in direct ratio to the amount of shock present as demonstrated by the clinical observation and cell nephelation.

As a result of these experiments it is possible to recognize accurately, by studying the various cells under the microscope, just what form of anæsthesia was used in each individual case. This does not mean that in every instance the deductions have been correct, but that the percentage of error has been very small. It can be truly said that the decision in regard to the kind of anæsthetic used must be based upon the clinical symptoms and bedside observations, or the microscopical study of the cellular nephelation; the latter would be the better method, as the degree of error shown by the tabulation was less than ten per cent.

In carrying out these experiments, the following anæsthetics have been used:

*I. General*

- a. Ether*
- b. Chloroform*
- c. Ethyl Chloride*
- d. Nitrous oxide*
- e. Nitrous oxide and oxygen*
- f. Nitrous oxide, oxygen and ether*

*II. Local*

- a. Novocaine*

*III. Spinal*

- a. Stovaine*

In each of these instances no narcotic was given previous to the anæsthetic, but, to augment the usefulness of the conclusions drawn from this, narcosis without any anæsthetic has also been used. Sometimes morphine alone, morphine and atropine or morphine and scopolamine were used.

In previous papers it has been shown that guinea pigs stand large doses of narcotic, often fully three times the amount required by a human being of normal weight.

Further, the same operation has been performed upon an animal which received no narcotic, anæsthetic or other supporting influence in order that, having the normal cells of the varied organismal structures of the guinea pig for one end control, an extreme end or degree control might be had by producing the same amount of pain nerve transmission unhampered by any supporting measures. In this way, the normal cells could be placed at one end and the cell of the non-supported operated pig at the other. By placing the structural studies of the cellular influence under the varied anæsthetic or narcotic influences between these two controls, more definite conclusion and truthful deductions could be drawn. In this way an unenthusiastic description of the experiments could be based upon conclusions drawn from true deductions.

Each experiment was done very carefully and with an extreme degree of deliberation. To cause a like amount of pain in each instance in a pig without any supporting measures, an operation in which the degree of sepsis played no part was chosen. There has

FIG. 11.



A



B

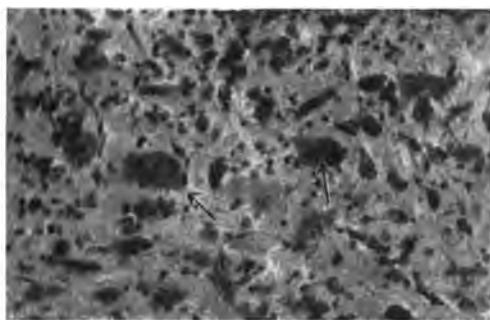


C

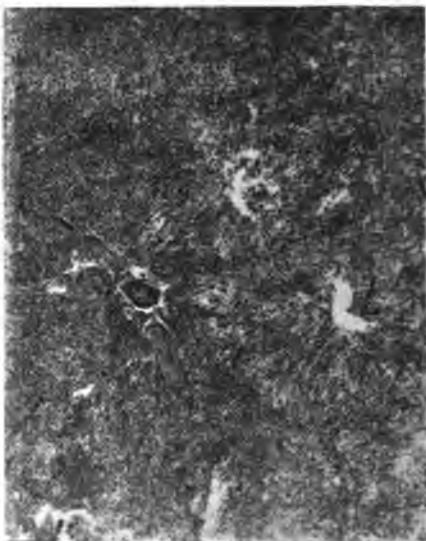
Guinea pig killed with lethal dose of morphine. No cellular change. A, spinal cord; B, striated muscle; C, liver.

FIG. 12.

*A*



*B*

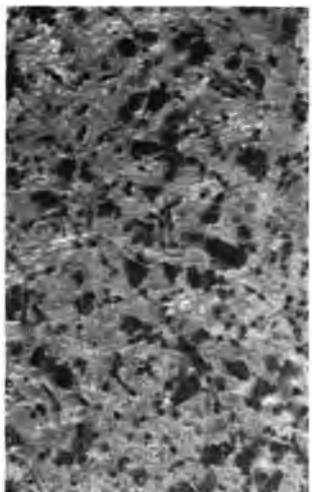


*C*

Guinea pig is given no narcotic, anaesthesia or other supporting measures. Note: Extreme degree of nephelation to marked vacuolation. Pain nerve transmission first of degree. *A*, cerebral cord; *B*, striated muscle; *C*, liver.

FIG. 13.

*A*



*B*



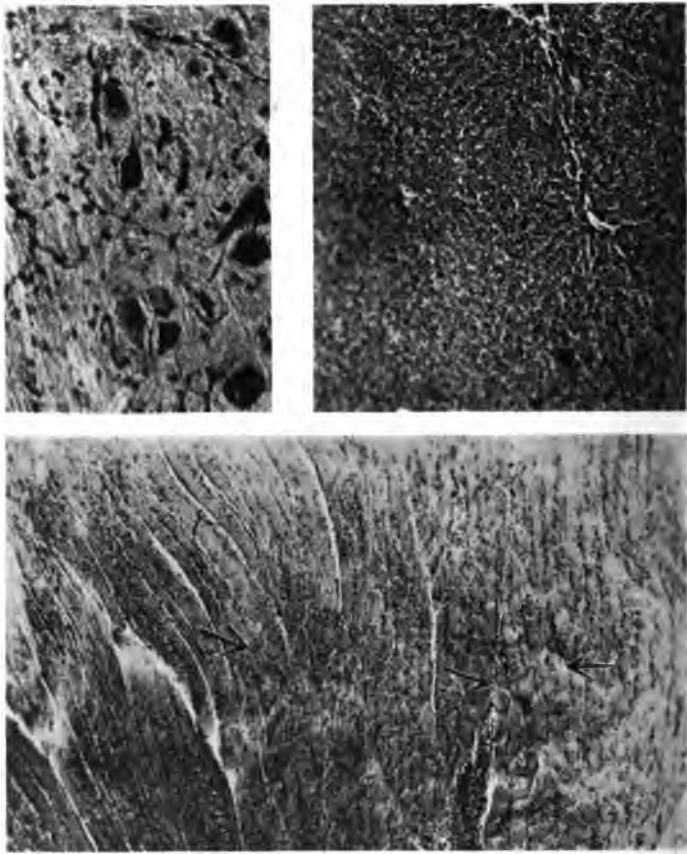
*C*

Guinea pig under ether anaesthesia. No other supporting measures. Note: Marked nephelation to point of vacuolation. Pain nerve transmission of first degree. *A*, cerebral cord; *B*, striated muscle; *C*, liver.

FIG. 14

*A*

*B*

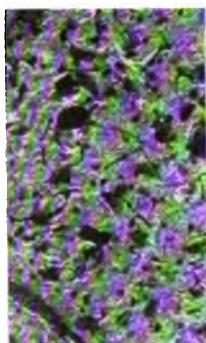


*C*

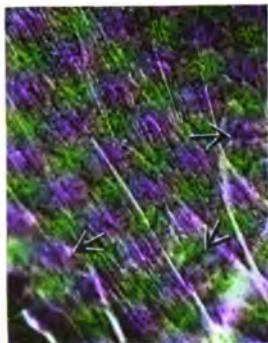
Guinea pig under chloroform anesthesia. No other supporting measures. Note: Marked nephelation to point of vacuolation. Pain nerve transmission of first degree. *A*, cerebral cortex; *B*, striated muscle; *C*, liver.

FIG. 15.

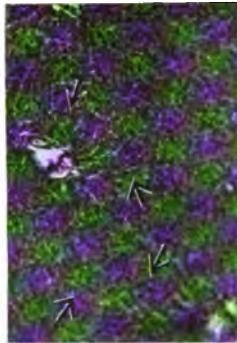
A



B



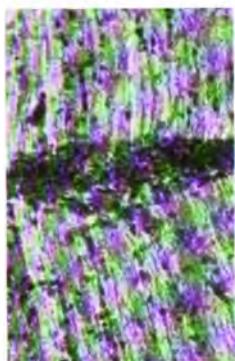
C



Guinea pig under ethyl-chloride anesthesia. No other supporting measures. Note: Marked nephelation to point of vacuolation. First degree pain nerve transmission. A, cerebral cortex; B, striated muscle; C, liver.

FIG. 16.

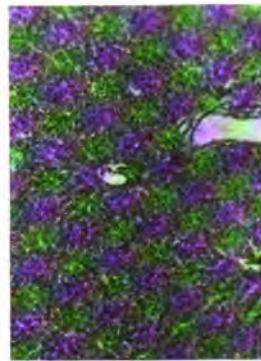
A



B



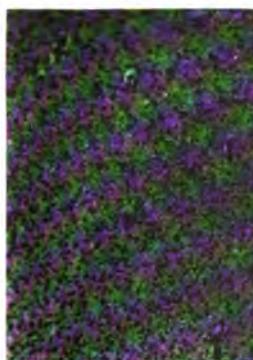
C



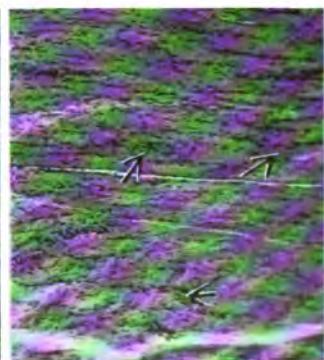
Guinea pig under nitrous-oxide anesthesia. No other supporting measures. Note: Marked degree of nephelation to point of vacuolation. First degree pain nerve transmission. A, cerebral cortex; B, striated muscle; C, liver.

FIG. 17.

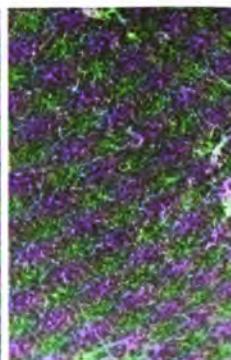
A



B



C



Guinea pig under nitrous-oxide-oxygen anesthesia. No other supporting measures. Standard operation. Note: Nephelation without vacuolation. Second degree pain nerve transmission.



been no break in continuity of skin or mucous membrane. In order to accomplish this result, the bones of a hind leg have been fractured and mutilated. In human beings this same procedure produces great pain and would warrant the conclusion a proper procedure to gain a just end had been selected. In each pig the bones of a hind leg were roughly broken and then as roughly rubbed together and mutilated for a ten minute period. Great care was taken to control this time absolutely.

The results of our findings are as follows:

*Experiment No. I.*—This experiment was done merely for control and comparison and the guinea pig was killed by a lethal dose of morphine without any operation. Fig. 11, *A. B. C.*, shows microscopical cellular study. Note that the cells are normal in size and construction, that no metabolic changes have taken place, and that there is no pain nerve transmission.

*Experiment No. II.*—This guinea pig was not given any narcotic or anæsthetic or other supporting measures. The standard operation was performed. Fig. 12, *A. B. C.*, shows marked changes in cord and cerebral cortex cells as well as in striated muscle and liver cells. Note the extreme degree of nephilation which extends even to the point of vacuole formation. The nerve pain transmission of first degree is very marked.

*Experiment No. III.*—Ether anæsthesia. Standard operation. Fig. 13, *A. B. C.*, shows marked degree of nephilation even to point of vacuolation. Pain nerve transmission of first degree is marked.

*Experiment No. IV.*—Chloroform anæsthesia. Standard operation. Fig. 14, *A. B. C.*, shows marked degree of nephilation, even to point of vacuolation and pain nerve transmission of the first degree.

*Experiment No. V.*—Ethyl-chloride anæsthesia. Standard operation. Fig. 15, *A. B. C.*, shows degree of nephilation even to the point of vacuolation and pain nerve transmission of first degree.

*Experiment No. VI.*—Nitrous-oxide anæsthesia. Standard operation. Fig. 16, *A. B. C.*, shows degree of nephilation even to point of vacuolation and pain nerve transmission of first degree.

*Experiment No. VII.*—Nitrous-oxide and oxygen anæsthesia. Standard operation. Fig. 17, *A. B. C.*, shows degree of nephilation without vacuolation but the pain nerve transmission of second degree.

*Experiment No. VIII.*—Nitrous-oxide-oxygen-ether anæsthesia.

Standard operation. Fig. 18, *A. B. C.*, shows degree of nephelation without vacuolation but the pain nerve transmission of second degree.

*Experiment No. IX.*—Novocaine infiltration, nerve block. Standard operation. Fig. 19, *A. B. C.*, shows discrete degree of nephelation. Cellular changes so sparse that fright could easily account for this minor change. The animal did not struggle in any way and apparently suffered no pain at all. The pain nerve transmission is of third degree.

*Experiment No. X.*—Stovaine in spinal canal. Standard operation. Fig. 20, *A. B. C.*, shows no nephelation or other cellular changes but pain nerve transmission of fourth degree.

By these experiments the degree of pain nerve transmission is clearly shown. For the purpose of classifying this pain nerve transmission an arbitrary scale of first, second, third and fourth degree has been used.

#### PAIN NERVE TRANSMISSION OF THE FIRST DEGREE

In this is included all cases showing confluent nephelation accompanied by vacuole formation, the most marked extreme of cellular changes found in these experiments.

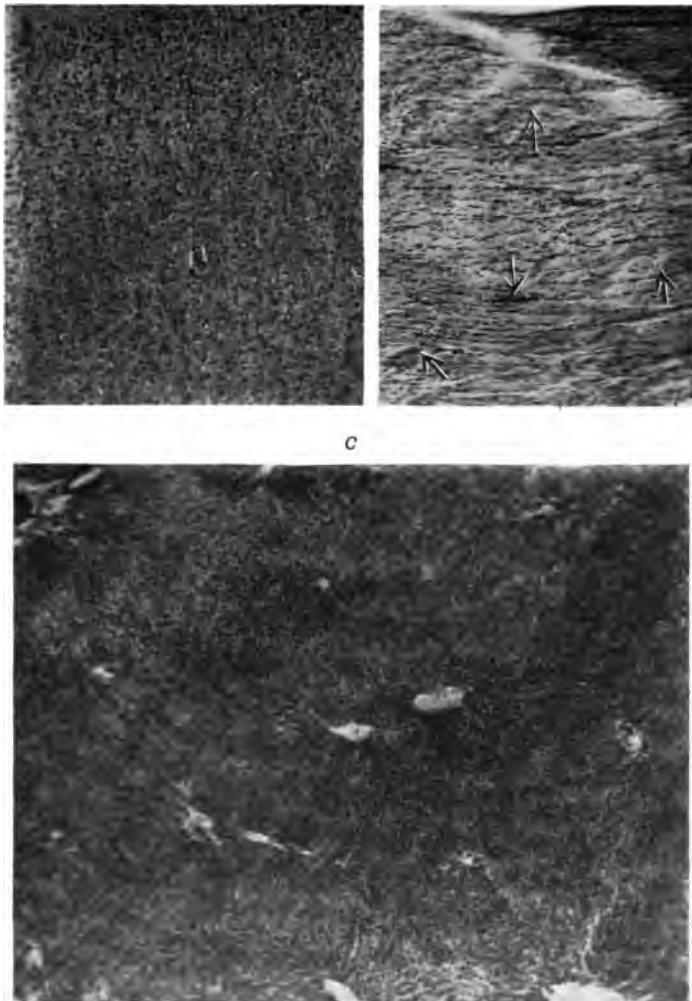
First degree changes were found in experiments Nos. II, III, IV, V and VI. Thus it is shown that confluent nephelation accompanied by vacuole formation is found in all cases where only a general anaesthetic was used. This clearly proves what Crile and Moynihan have previously stated that a general anaesthetic merely produces unconsciousness and eliminates fright. In no way does the anaesthetic control pain nerve transmission. In other words merely because a patient is so conditioned that he cannot react to the sensation of pain does not mean that the pain is absent. These experiments clearly show that pain is transmitted just as much when a patient is under the influence of a general anaesthetic as if they were wide awake. Again it goes to show that fright plays only a small part, since its influence is not recognized when some form of nerve block is not used.

Fright transmission influence travels along different fibres from pain transmission with similar result but less in degree. This degree is so much less that its influence does not appear when pain transmission is marked. In other words, the pain entirely predominates

FIG. 18.

A

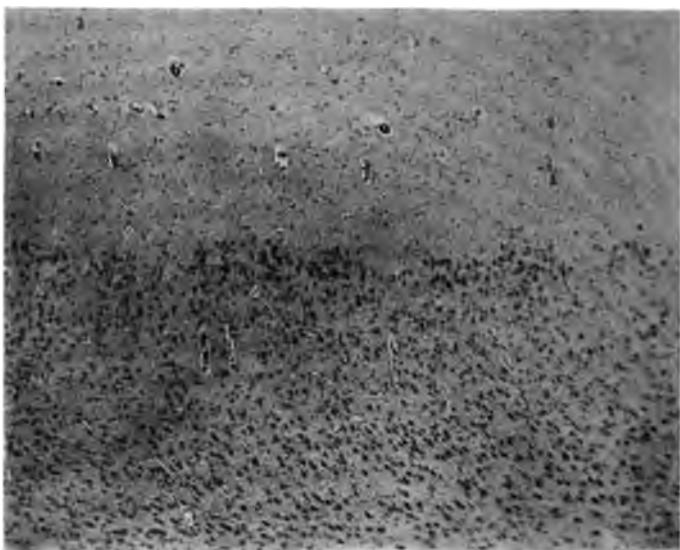
B



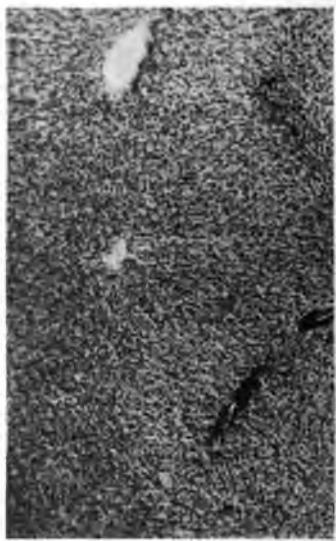
Guinea pig under nitrous-oxide-oxygen-ether anesthesia. No other supporting measures. Standard operation. Note: Nephelation without vacuolation. Second degree pain nerve transmission. A, cerebral cortex; B, striated muscle; C, liver.

FIG. 19

A



B

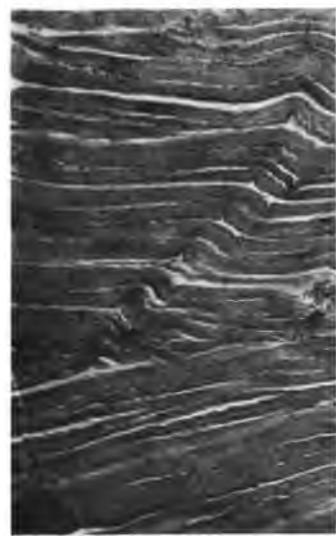


C

Guinea pig. Novocaine infiltration, nerve block. No general anesthetic or other supporting measures. Standard operation. Note: Discrete nephelation. Third degree pain nerve transmission.  
A, cerebral cortex; B, striated muscle; C, liver.

FIG. 20.

**A**



**B**



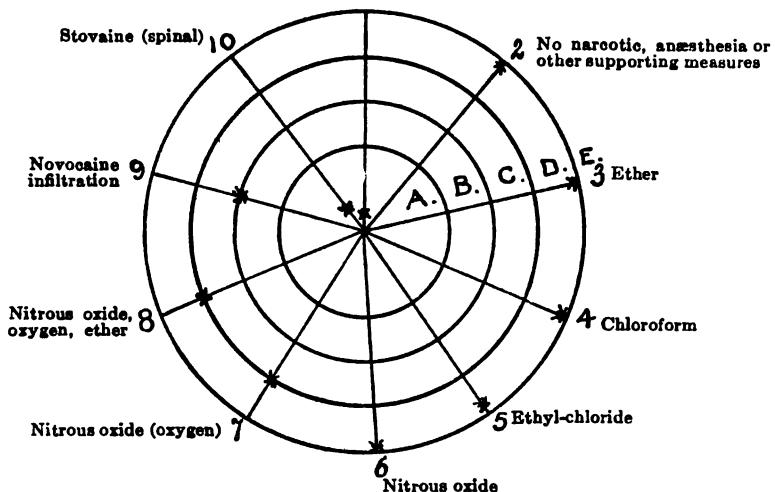
**C**

Guinea pig. Stovaine in spinal canal. No general anaesthetic or other supporting measures. Standard operation. Note: No cellular change. Fourth (nil) degree pain nerve transmission. **A**, cerebral cortex; **B**, striated muscle; **C**, liver.



and the fright influence is lost in the shuffle. If you will but compare Experiment No. II with the other experiments which show the first degree pain nerve transmission, this point will be clearly brought out. If pain transmission is great, the influence brought to bear and damage done is just as great whether the patient be conscious or unconscious if factors are equal. The result clearly proves that without other supporting influences a general anæsthetic does no

FIG. 21.  
Lethal dose of morphine

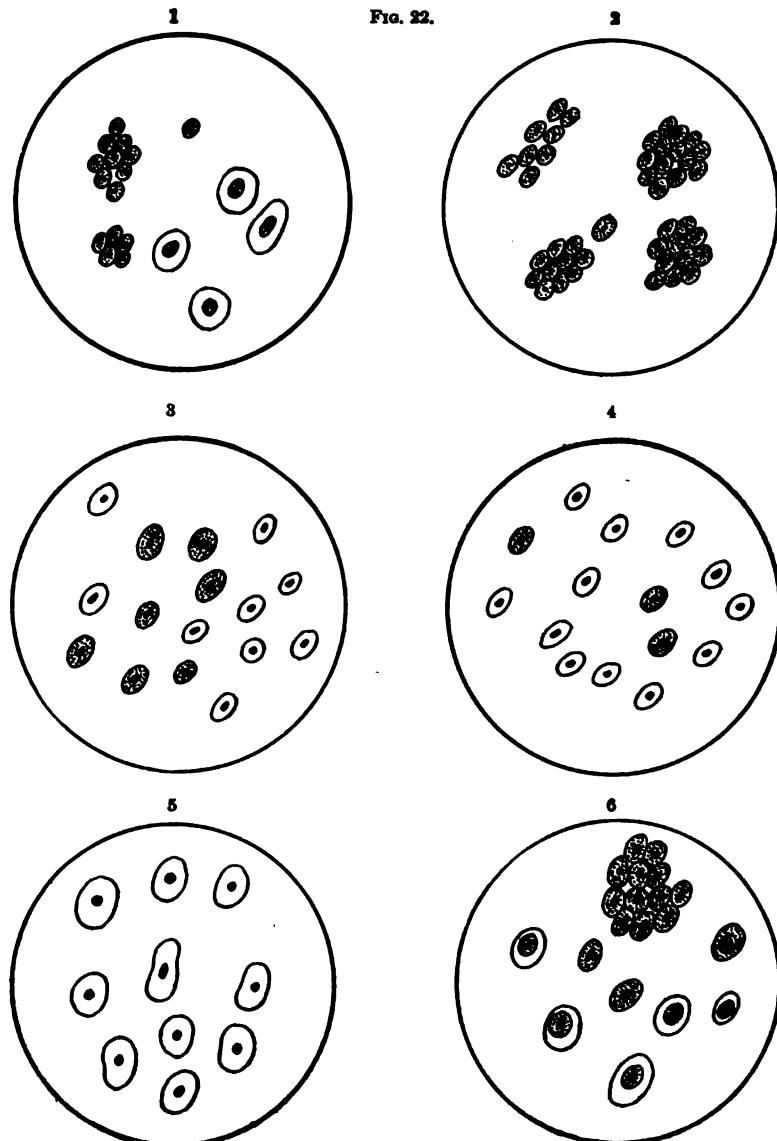


Cobweb construction to show direct ratio between pain nerve transmission and the anæsthetic used. Key: *A*, pain; *B*, fourth degree; *C*, third degree; *D*, second degree; *E*, first degree. \* Marks degree of pain nerve transmission. Numbers indicate number of experiment. Each degree of pain nerve transmission shown.

more than to eliminate fright, produce unconsciousness and give perfect muscular relaxation. More than a general anæsthetic is necessary if the eliminating of surgical shock with concomitant after effects is desired.

#### PAIN NERVE TRANSMISSION OF THE SECOND DEGREE

In this class are placed arbitrarily all cases showing confluent nephelation but no vacuole formation. Pain nerve transmission has been lessened as far as the elimination of vacuolation. In other words, the pain transmission has been enough to produce confluent nephelation but these cells have not vacuolated and are still able to partially or wholly recover after a time.



1. First degree pain nerve transmission.  
Confluent nephelation and vacuolation.
2. Second degree pain nerve transmission.  
Confluent nephelation; no vacuolation.
3. Third degree pain nerve transmission.  
Discrete nephelation only.
4. Fourth degree pain nerve transmission.  
Nearly all cells normal. Occasionally partial nephelation.
5. First end (normal cell) control.
6. Further end (operation without any supporting measures) control.

The degrees of cellular alteration between the two extremes are shown in the previous illustrations.

This degree of pain nerve transmission is shown in Experiments Nos. VII and VIII, where discrete nephelation is found. No agglutination of nephelated cells is found and the depth of nephelation is not so marked. Border cells seem most marked while only a few of the more central cells are involved at all.

Strange as it may seem, without other supporting measures, only a general anæsthetic was used in these two experiments, which would seem to cause pain nerve transmission of the first degree. Some extraneous influence was inaugurated which prevented confluent nephelation, vacuolation, agglutination of the nephelated cells, and resulted in a more central nucleal involvement with the depth of nephelation lessened. What was this influence? After a most pains-taking study the conclusion was reached that as oxygen had been used in conjunction with the general anæsthetic this element must have some influence in favoring a lessening of the degree of pain nerve transmission. Just what influence oxygen has cannot be stated at the present time, but later it may be possible to explain this elemental influence. By the process of exclusion the credit seems to belong to oxygen. In some manner, not yet determined, oxygen plays a cellular conservation part. Whether this influence is due to the offsetting, stimulating influence of the presence of oxygen, whether it is due to some chemical union taking place which supports the cells, or whether fluid saturation is conserved to a greater degree by the oxygen, cannot be definitely stated. For the present it can be proved that when oxygen is present in conjunction with some general anæsthetic the amount of cellular damage and destruction is greatly lessened. Pain nerve transmission of the second degree is apparently under the control of oxygen.

#### PAIN NERVE TRANSMISSION OF THE THIRD DEGREE

In this class we have placed those cases that show only discrete nephelation. This nephelation is very small. The nephelated cells are not as large and most of them are periphery in location while the nephelation itself is not symmetrical in so far as regularity of change is concerned. In fact, it would seem, by looking at the photographs from Experiment No. IX, that there should have been no pain. The writer believes that this was true and that the slight amount of nephelation comes from fright influence rather than nerve

pain transmission. The animal remained perfectly quiet during the experiment and showed no outward signs of pain.

In previous experiments it has been shown that no cellular changes took place when nerve block and a general anæsthetic were used coöperatively. It has also been clearly shown that the giving of any general anæsthetic merely produces unconsciousness and thus eliminates fright. For this reason it is safe to assume that what nephelation was present in this degree was due to fright alone.

#### PAIN NERVE TRANSMISSION OF THE FOURTH DEGREE

Under this class has been placed those experiments from which no nephelation resulted, and therefore, there was no pain. Experiment No. X illustrates this very well. Stovaine was given in the spinal canal. Within ten minutes the parts below the point of injection were completely numbed. No response from pain could be elicited. These illustrations show how completely pain nerve transmission was blocked. Note how clear the cells are and how firm the whole field appears.

#### CONCLUSIONS

1. Pain nerve transmission produces the condition known as surgical shock.
2. The nephelation produced is in direct ratio to the pain nerve transmission and in inverse ratio to the degree of nerve block.
3. Any general anæsthetic merely produces unconsciousness and does away with fright.
4. The degree of pain nerve transmission is not controlled to any degree by a general anæsthetic.
5. Patients operated upon under local analgesia do better than under a general anæsthetic.
6. The combined methods of general anæsthesia and nerve block produce the best results.
7. Fright should be eliminated as well as pain nerve transmission if the best results are to be expected.
8. Fright alone can produce a certain degree of surgical shock.
9. When nerve block is complete the element of surgical shock is eliminated and no matter how long the operation is, the work can be done safely.

## THE OPERATIVE TREATMENT OF CHRONIC POLIOENCEPHALITIS SUPERIOR\*

By CECIL E. REYNOLDS, M.R.C.S., ENG.

DURING the past few years in Los Angeles I have operated upon seven children for varying combinations of imbecility, paralysis and fits having signs of cortical compression or irritation. Cases due to syphilis or hemorrhage are not included in this series.

In four of these cases I was able to demonstrate a watery condition of the pia arachnoid covering the affected cortex, one of these, viz.: the case presented at this meeting, has been cured and this case and two others had an acute onset after the first year of birth.

I will give a short account of all seven cases.

The first case was a girl of eleven years who showed progressive imbecility and a most vicious disposition since an attack of headache and vomiting at the age of two, which recurred at intervals for several years.

She presented signs of a slight cerebral diplegia and had convergent strabismus. I performed a craniectomy without opening the dura and made new sutures one half inch wide in both coronal and sagittal planes. A very considerable improvement in disposition and intelligence resulted and the strabismus was cured; but owing to the limited surgical procedure this case is of little value in the present discussion. A full account of it was published in the *Southern California Practitioner*, May, 1914.

The second case was referred by Dr. F. L. Anton, and this same operation performed. The child was four years old, was very diplegic and could not stand at all; but this time I opened a small piece of dura mater and found a watery condition of the pia arachnoid. No improvement resulted.

I adopted a different procedure on the next case, referred by Dr. John Ferbert, a boy of seven years of age who had been a very bright

---

\* Paper read and successful case shown before the Los Angeles County Medical Association, June 6, 1918.

and healthy child up to the age of four years, when he had an acute febrile illness, after which he gradually lost the power of speech and became a diplegic idiot.

In this case I turned down a large osteoplastic flap over the left Rolandic area and on opening the dura found the same watery condition of the pia arachnoid as in the previous case. I stitched back the dura, replaced the flap and performed a permanent subtemporal decompression.

He made quite appreciable progress for a while but about three months later the limbs on the left side presented signs of increasing paresis, so I turned down a flap over the right Rolandic area and finding the same condition of water bed over the right cortex I attempted to evacuate the fluid. The removal of the fluid was followed by immediate shock as I have found to be the case also when syphilitic cysts are evacuated in children, and he died shortly after.

The next case was a very bright girl of eleven years who had suffered from Jacksonian epilepsy since the age of four years. The fits began in the left thumb and then "marched" in orthodox fashion. Upon turning down an osteoplastic flap over the Rolandic area I found no abnormal condition of the cortex; I decided not to excise the thumb centre, and replaced everything in *statu quo*.

I believe that her ultimate complete recovery resulted from the following accident. That night she had reactionary hemorrhage of the middle meningeal artery, which I am afraid is almost indefensible, although everything looked secure when closed up. By the time I had religatured the vessel and cleansed everything thoroughly she was too much shocked to allow of the dura being stitched in place. Accordingly I simply laid it back over the cortex and replaced the bone flap.

She was paretic in the arm for a few days, but this quickly cleared up and she is now quite well. In the light of subsequent events I attribute her cure to the complete decompression of the dural sac.

Fig. 1 is that of another imbecile child in one of his almost continual fits, upon whom I did a similar exploration and found the same condition of boggy pia arachnoid already referred to. In this case I stitched back the dura over the Rolandic area and left a permanent subtemporal decompression with very slight improvement.

Case of polioencephalitis superior in a fit.



FIG. 1.



FIG. 2.

FIG. 3.



A large osteoplastic flap was turned down over the whole left Rolandic area and on reflecting the dura the same condition of fluid was seen in the meshes of the pia arachnoid, exactly as in the cases just now described so that the cortex appeared as if looked at under glass or through a jelly.

Having learned the danger of freely opening the arachnoid and evacuating the fluid, which is remarkable as a rapid shock producer, I merely made some little pin pricks in the membranes and laid the dura back upon them without stitching and replacing the bone and scalp.

Following the operation she had six convulsions during the first day, mainly on the left side of the body.

Next day she had twenty-five convulsions on the left side, one on the right side and two of the whole body.

Next day (November 11) she had nine convulsions up to 10 a. m., after which she sat up in bed and started playing and has never had another fit since.

The only additional treatment was started this day and consisted of urotropin grs. ii (hexamethylamine) t.d.s., which she is still taking at intervals and she also had for six days only hydrarg. cum cret. gr. ii t.d.s. as a laxative.

She is now, as you see, perfectly well mentally and physically and a good talker.

In all of these seven cases syphilis was carefully excluded. All but one (Case II, Dr. Anton's case) were exceptionally fine and healthy babies until the second year of life. Most of them had a definite acute onset and several were given urotropin for varying periods after operation.

I believe that the condition found is the result of an infection of the cortex of the same nature as poliomyelitis owing to its local distribution; but the meningococcus cannot be excluded until lumbar puncture is regularly performed in the acute stages of apparently causeless convulsions.

There is one other point worth mentioning in regard to the surgery and that is the question of regeneration of the dura. I found in one of a series of cases in which I left the dura unstitched and in which the child died several months later from another cause, the autopsy

FIG. 4.



Piece of dura mater removed several months after operation, showing where new dura has grown from line of demarcation. A, B, and C shows edge of the old dura. New dura has broken away from upper edge post-mortem.

FIG. 5.



FIG. 6.



Pial aspect of dura shown in Fig. 4. Pia mater is thrown into folds on both old and new dura. Line of junction between new and old dura as shown by transmitted light. Photographed from pial aspect.

revealed the fact that a new dura had grown from the retracted edge of my dural flap up to the rest of the dura. The new dura was intimately connected with the pia arachnoid beneath but had caused no symptoms of irritation any more than in the two successful cases cited and thus the operation had enlarged the whole dural cavity and Nature had restored the protective covering to a nearly perfect state. Figs. 4, 5, and 6, demonstrate this.

I feel justified in concluding from these results that the sequelæ of polioencephalitis superior can be greatly benefited at any stage short of the hopeless idiot by placing the sodden pia arachnoid in a condition where it can slowly drain into the lymphatics and at the same time allow the brain to expand. In order to do this it is necessary to freely open the dura mater and leave it open.

I am also strongly in favor of the prolonged use of urotropin on general principles at all stages of the disease.

Microscopic sections from Case III have been prepared for me through the kindness of Dr. Levin and Dr. Bolton of the Pacific Wassermann Laboratories, and they show some leucocytic infiltration of the dura mater and slight vacuolation of the outer layers of the cortex.

## FRACTURES OF THE FUNDUS OF THE COTYLOID CAVITY WITH INTRAPELVIC DISLOCATION OF THE HEAD OF THE FEMUR

By MAURICE MARQUEZ, M.D.

Montpellier, France

**CASE.**—Mrs. S., aet., thirty-eight years, fell from a carriage, striking on the left hip. She could not walk and complained of severe pain in the knee. The patient was seen about 18 hours after the accident. The patient is a strong woman and has never been ill.

She lay on the back with the lower limbs extended. The left limb is in adduction and outward rotation. The external border of the foot almost touches the plane of the bed. The limb appears notably shortened and measurements showed a shortening of about 4.5 centimeters.

The region of the trochanter is flattened and depressed; the upper border of this apophysis, which is higher than normal, is consequently nearer the iliac crest. Transversal pressure over the great trochanter and from below upwards on the knee produces pain which the patient locates deeply in the pelvis. This pain is also provoked by pressure over the crural arch and towards the lower portion of the internal iliac fossa.

The limb can be flexed on the pelvis and brought into abduction; but abduction and external rotation are impossible. All these movements are very painful. Crepitation was never detected.

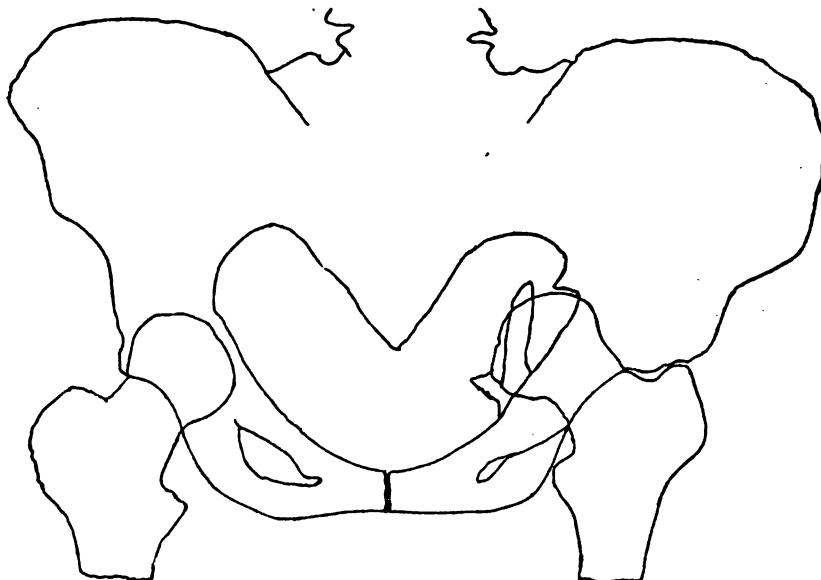
A fracture of the femoral neck was made, with the reserve of what might be found by radiography as soon as the patient could be moved. Meanwhile, the limb was placed in a grooved wire splint with continued extension.

A radiography was taken ten days later (see Fig. 1), and revealed the mistake in diagnosis. The case was one of fracture of the cotyloid cavity without any lesion of the femur. As seen in the diagram the fundus of the cotyloid cavity is broken into several fragments which project into the pelvic cavity. The contours of the foramen ovale

appear intact. It is by this opening of the cotyloid cavity that the head of the femur has penetrated the pelvis while the neck has become placed in the fractured contour. It will also be noted that as a consequence of these lesions the advance of the trochanters is towards the pelvic sides and their excursion towards the iliac crest.

Vaginal examination was made and showed that on the left lateral wall of the pelvic enclosure there is a distinct spheroidal projection

FIG. 1.



Fracture of the fundus of the left cotyloid cavity without fracture of the femoral neck. Drawing made from a radiograph of the writer's case.

represented by the head of the femur, because movements transmitted to the femur or pressure made on this bone are detected by the exploring finger.

By pressing on this projection severe pain is produced. Six months later another examination revealed the same projection, but more distinctly, because at this time the swelling which existed before had disappeared. We also assured ourselves that the maximum pain was seated deeply in the left part of the pelvis. Fruitless efforts at reduction were made, because it certainly seemed that if strong traction were made the femoral head might be disengaged and lowered,

but instead, the head went upwards every time that traction was stopped and as these manœuvres were very painful they were not continued, the limb being simply left in the grooved wire splint with continuous extension.

On the twenty-fifth day the patient began to get up and took a few steps with the help of crutches. Movement of the limb was very painful and this lasted for some time. Even at rest the patient suffered, complaining of a persistent gonyalgia.

At present the pain has almost completely disappeared, but there remains a considerable limp as there is about 4 centimeters' shortening and a marked scoliosis has developed. Flexion of the thigh is fairly good although not as extensive as on the right. On the other hand, abduction is absolutely impossible.

Vaginal examination still reveals its projection due to the fracture, and it seems to us that a partial or even total bony consolidation has taken place, because the movements of the femur are no longer perceptible to the exploring finger.

The frequency of fracture of the cotyloid cavity with inward depression of the fragments is certainly greater than is generally supposed, if one simply takes into consideration the number of recorded cases. This is also the opinion of Hamilton, Gay and Ollier and as further proof I may add the statistics of Groessner. He was able to collect in Bardenheuer's service within the space of nine months 362 cases of fracture, 10 of which involved the pelvis and only 12 of the femoral neck. Now, among the 10 cases involving the pelvis, 4 were distinctly fractures of the cotyloid cavity with depression of the fragments.

As to the age at which this fracture preferably occurs, one can distinctly fix the extreme limits after statistics made in this direction by Ohandjanian, Virevaux and Guibé. They all agree that the maximum of frequency is between the ages of ten to thirty years, and this seems easy to explain. In the first place, from the age of ten to twenty years, the frequency of this lesion is not surprising, because it is between the sixteenth and eighteenth years of life only, that the three bones forming the coxal bone are completely united, while the femoral neck presents the best conditions of resistance.

It is certainly between the ages of twenty to thirty years that man, in full possession of his strength and vigor, indulges in the hardest labor and the more perilous exercises; in a word, it is during this decade that his life is more exposed to trauma. But man still retains his physical activity for years after the age of thirty, so that the frequency of intrapelvic dislocations of the femoral head, without reaching the preceding maximum, is still considerable. This is proven by the statistics of Ohandjanian and Guibé. The former found 12 cases occurring between the ages of thirty to sixty years and 14 cases between the ages of ten to thirty years, while Guibé's tables give 9 cases between the ages of thirty to sixty years and 11 cases between the ages of ten to thirty years.

On the contrary, as soon as the age of sixty years is passed, or before the end of the first decade of life, this particular fracture is an exception. In elderly subjects the particularly rapid rarefaction of the bony tissue of the femoral neck results in its being the weakest point of the skeleton of the lower limbs, while clinically we know that fractures of the femoral neck are common enough. In the very young, the rarity of these fractures is explained by the nature of the way of living and by the very great elasticity of the bony pelvis.

The influence of sex in the production of the fracture under discussion is, in itself, absolutely unimportant. However, I am led to believe that, given a trauma of equal severity, an intrapelvic dislocation of the head of the femur is much easier to produce in the female than in the male. In the former, the particular oblique position of the coxal bone, on account of the physiological adaptation of the pelvis, gives the femoral neck an almost normal direction to the plane of the fundus of the acetabulum, while in the male the direction is distinctly oblique from below upwards. I shall soon endeavor to show all the importance that this direction may have in the production of these fractures.

Among the traumatisms capable of producing this fracture there are three principal ones, namely, (1) a fall or blow on the great trochanter, (2) a heavy weight falling on the shoulders and (3) a fall on the feet.

In the immense majority of cases of intrapelvic dislocation of the head of the femur, resulting from a fall or blow on the great tro-

chanter, the fracture of the cotyloid cavity usually results from a direct fall on the hip, the height of the fall varying from one case to another. However, a violent blow on the hip may also produce a depressed fracture of the acetabulum. For that matter the type of traumatism is of little import, since these fractures can occur only when certain conditions are fulfilled.

In the first place, the traumatism must be a violent one with a very resisting femoral neck, otherwise the latter would be the first to give way. And lastly, but not least, there must be a particular direction of the traumatic force. In fractures under consideration, the neck of the femur represents this direction, since it is by it that the pressure resulting from the trauma is directly transmitted to the cotyloid cavity. The best position for the limb to be in, in order that the neck of the femur shall perforate the fundus of the cotyloid cavity, is when the femoral neck presents a normal position in relation to the plane of the acetabulum.

When these conditions are not realized at the time the head of the femur is forced violently against the acetabulum, it would come obliquely upon the plane of the latter and would be, so to speak, reflected and have a tendency to dislocation rather than otherwise. Perhaps, striking against the brim of the acetabulum, it would be stopped and then one of two things must happen. Either the brim of the acetabulum will resist and there is fracture of the femoral neck from bending or a portion of the rim of the acetabulum will be torn away by a very resistant femoral neck. In other cases, the head of the femur continuing to slide in the cavity, tears the capsule, becoming completely dislocated, so that perforation of the cavity becomes possible.

The direction of the femoral neck, which must be normal to the plane of the fundus of the cotyloid cavity in order that a depressed fracture shall occur, will be realized whenever the lower limb is in a normal position or even in a slight abduction with outward rotation. By this double movement the great trochanter is carried upwards and backwards while the head of the femur effects just the inverse movement (downwards and forwards), and the axis of the femoral neck will be found almost in a plane perpendicular to that of the acetabulum.

But these conditions are fulfilled in only a very restricted number of traumatisms and this explains the infrequency of these fractures. It is even quite a difficult matter to produce a depressed fracture of the cotyloid cavity experimentally, since out of a total of 17 experiments on the cadaver, Virevaux succeeded only once in producing an intrapelvic dislocation of the hip, while Guibé was invariably unsuccessful and Thévenot obtained only positive results by replacing the femur by a box-wood stick.

When a subject falls upon the feet, which results in a depressed fracture of the cotyloid cavity, the neck of the femur must be short and thick with a very resisting diaphysis, while the limb must be in marked abduction, so that the shock is transmitted normally to the cotyloid cavity. This is in reality, a fracture by "*contre coup*," similar to those met with at the base of the skull following a similar fall. In the case of the acetabulum I know of but one authentic instance, this being published by Kroenlein.

There is only one authentic case of depressed fracture of the cotyloid cavity resulting from the fall of a heavy weight on the shoulders. It was that of a mason who received a blow from a sack of plaster which fell from the third story of a house, striking him on the *left* shoulder, which resulted in a fracture of the *right* cotyloid cavity.

*Symptomatology.*—The history of the accident will usually be of little avail in making the diagnosis. The patient will generally have sustained a fall from a certain height, striking on the hip, or a violent shock will have been received over the coxofemoral joint. He was not able to get up after the accident. The mental faculties are ordinarily preserved at the time of the receipt of the injury, but shock or coma may be present.

Among the functional disturbances the first to consider are pain and impotency of the limb involved. Pain is very sharp, spontaneous and increased by the slightest contact over the site of the injury. But it offers a very particular characteristic, namely, that the patient himself locates the maximum point. In reality, the patient suffers greatly and he will tell you that it is *within the abdomen* that the pain is most severe. Pressure made over the crural arch and above it at the level of the lower part of the internal iliac fossa will cause the patient to cry out. Another particular character of the pain

that we have not found mentioned in the reports of other cases, but which was very marked in our cases, is the pain complained of in the knee. This pain was very persistent, since the patient still complained of it twenty-five days after the accident.

By vaginal or rectal examination the site of the pain can be exactly located.

Functional impotency is usually complete, although from the nature of the lesions it is, in reality, only partial. This is evident, because in every fracture of the cotyloid cavity with penetration of the head of the femur into the pelvis, the femoral lever remains intact as well as the muscular masses which move it. There is simply a displacement of the point of application of resistance, which is represented by the body weight and transmitted to the femur by the acetabulum. Now, although the fundus of the cotyloid cavity has to a certain extent disappeared, the solidity of the rim of the acetabulum is in no way diminished, so that the femur will resist all pressure produced on it by the trunk, whether this pressure is applied to its head or neck.

Therefore, I believe that the upright position is possible, and this was so in the case I report. The movements of flexion and extension of the thigh on the trunk should be normal; abduction and adduction limited either because the upper border hits against the rim of the acetabulum during abduction or that the head of the femur becomes caught by its upper portion by the sharp elevation of the bony brim fractured in the adduction. Alone internal or external rotation of the limb seems to me impossible, the neck of the femur being caught in the circumference of the line of the fracture which prevents it from going forward or backward, according to the direction of the rotation. Briefly, I consider that the total functional impotency is simply the result of the violent pain.

There are certain physical signs which at once draw the attention. In the case here reported the limb on the injured side was extended on the plane of the bed in adduction and outward rotation, while in some of the reported cases it was found in abduction and outward rotation while others present a flexion of the leg on the thigh. The hip is considerably flattened or even depressed; the limb appears markedly shortened and this is, in reality, the case. There may be

an ecchymosis above the ligament of Fallopius, a symptom which Lössen considered as pathognomonic of these fractures. This symptom was absent in my case and Guibé, François and Kontorowitch have never met with it.

By palpation, the depression of the trochanteric region is distinctly felt. The great trochanter seems to have disappeared and it is detected only in the shape of a deeply seated marked projection, and appears to form one body with the pelvic belt. Its upper border has undergone a very notable ascension and to this ascension corresponds a real shortening of the limb of  $4\frac{1}{2}$  centimeters, as in my case, or even more. In Boeckel's case it was 5 centimeters.

Pressure made over this area will cause deep-seated pain, but crepitation cannot be made out.

Passive movements are all diminished in amplitude and cause such suffering that it is hardly humane to push this part of the examination very far unless an anaesthetic be given. All these symptoms do not point to a firm diagnosis and one should never neglect to complete the examination by very careful rectal or vaginal exploration.

By this means symptoms of capital importance are found. These are in the first place a pain localized in the region of the lesion, a spheroidal projection which is nothing less than the head of the femur in intrapelvic dislocation, since movements imparted to the lower limb are transmitted to the projection and are distinctly felt by the finger in the rectum or vagina. In some cases crepitation can be detected and Roux mentions a case in which the splinters of bone gave the sound of bits of porcelain shaken together.

If rectal or vaginal examination be made at the time of the first examination of the patient, a very large haemorrhagic collection will be discovered in the majority of cases and this haematoma may, to a certain extent, interfere with the perception of the above-mentioned signs.

A radiograph will give a very correct idea of the extent of the bony lesions.

One might, *a priori*, assume that all fractures of the acetabulum with penetration of the head of the femur into the pelvis should have a fatal evolution towards permanent ankylosis, unless an appropriate treatment is given in time to put a stop to its progress, but I hasten to add that this treatment is as yet to be found.

Among the unfortunate complications internal hemorrhage must be mentioned, due to a wound of the external iliac vessels from a splinter of bone. Another is a septicæmia resulting from suppuration of the intrapelvic haematoma (its nearness to the vagina or rectum makes such an infection easy); and lastly, there is pneumonia which occurs in any type of severe traumatic injury.

Such are the immediate complications which may result in death. As late complications it should be recalled that tuberculous arthritis is often a consequence of severe injuries to the hip joint. Guibé mentions a case of neuralgia of the obturator nerve following a lesion resulting from the fracture and Partridge relates the case of a woman in whom, following a depressed fracture of the fundus of the cotyloid cavity, the head of the femur, being badly reduced, became the cause of dystocia, requiring the application of the forceps.

As to the prognosis, it should always be reserved, at least during the early days, while one of the immediate complications is still possible. Later on it is good as far as the general health of the subject is concerned, but it should always be impressed upon the patients that, no matter how quickly a cure may result, there will always be difficulty in the movements of the limb on the injured side and that a permanent limp is almost sure to ensue.

From the fact that the symptomatology of depressed fracture of the fundus of cotyloid cavity is so precise, it would seem that the diagnosis should be readily made. However, the numerous mistakes made by most competent clinicians eloquently prove that such is not always the case. Therefore, when one is called to attend a fracture simulating that of the cotyloid cavity, the attention should just be directed to the hip in order to ascertain if the case is one of fracture of the neck of the femur.

The frequency of this fracture, the resulting pain, functional impotency, flattening of the hip and shortening of the limb would seem enough upon which to make a diagnosis. And nevertheless, by a more detailed examination, one quickly sees the error when the acetabulum is involved. The site of pain is deeper seated in the latter than when the lesion is in the femoral neck, the patient can stand and crepitation is absent. Then by rectal or vaginal examination, which should never be neglected in any case, the examining finger will

detect a rounded projection over the internal aspect of the cotyloid cavity, which will be recognized as the head of the femur by resorting to the means I have already mentioned. Also, pressure made on this projection is very painful, while around it one can feel splinters of bone which have been torn from the acetabulum. Nothing of the kind occurs in fracture of the neck of the femur, the internal aspect of the pelvis being perfectly normal.

When the rounded projection is felt it may be questioned whether or not it is a neoplasm of the pelvic wall or of the organs contained within it. But here again, rectal examination will remove all hesitation, because movements imparted to the limb on the injured side will be transmitted to the supposed tumor and distinctly felt by the finger.

There still remains one other pathological condition to be considered, namely, a forward obturator dislocation of the femur, but in this case nothing is felt per rectum and the member on the injured side is in marked outward rotation *without* shortening, while careful palpation will always detect the head of the femur outside of the pelvic enclosure.

It would seem from all that has been said that rectal or vaginal examination would in itself be sufficient to settle the diagnosis. However, during the first few days following the injury a very large haematoma may very easily hide those signs which should be obtained by rectal exploration, and it is in just these cases that radiography should be resorted to.

The want of autopsies and the scarcity of radiographs of this lesion make it difficult to give the pathology in any detail. The haematoma is evidently the consequence of the injury of some vessel from the fragments of bone and I know of instances in which the vessel involved was the obturator artery, gluteal artery and the external iliac vein.

The capsule of the hip joint may be completely ruptured or it may remain intact, while the round ligament is often ruptured, either at its femoral end or at its point of insertion in the cotyloid cavity, which may be torn away.

Such are the lesions met with at the time of the injury. Let us now consider how the process of repair takes place when these

fractures tend towards recovery. In these cases collected by Guibé the head of the femur was completely surrounded by a true bony shell, excepting at one point, the size of a quarter of a dollar, where it was fibrous in nature.

In the case here reported this same process probably took place, since at the present time the projection of the head of the femur is still perceptible *per vaginam*, but movements of the femur are no longer felt by the exploring finger. It is probable that a process of regeneration occurred from the periosteum.

It would seem indicated, considering the type of the lesions and displacement of the head of the femur, to treat these fractures like a dislocation, and in reality, the first thing to do is to make an attempt at reduction and then to find some means by which the reduction, if obtained, can be maintained.

When there is intrapelvic protrusion of the head of the femur through the acetabulum reduction is generally possible, but in some cases the neck may be so considerably engaged that it becomes impossible to reduce without danger of producing serious disturbances.

To reduce the dislocation an assistant, who must be very strong, holds the limb in extension while another assistant makes counter-extension by solidly immobilizing the pelvis, during which the surgeon supervises the effect of the traction by a finger introduced into the vagina. He will feel the special projection, formed by the head of the femur, disappear little by little and become imperceptible, indicating that reduction has been accomplished.

Continued extension in the axis of the limb offers the advantage of early mobilization, so that ankylosis can be avoided—the production of this complication being sure to occur if one puts the pelvis and lower limb in a plaster cast. Although continued extension certainly produces only an incomplete reduction (the tonicity of the gluteal muscles offer a resistance which is difficult to overcome), I prefer it to any other mode of treatment, particularly because it is within the reach of everybody and gives quite as good ultimate results, and because I esteem that a limp is far less serious than an ankylosis of the hip joint.

To continued extension in the axis of the limb, Rouz of Lausanne adds lateral traction. He says that if this is begun at once, not

only does it reduce the dislocation of the head of the femur, but the fragments of the cotyloid cavity as well, as these are drawn upon by the capsule which is inserted on them, so that at the same time that the head of the femur is withdrawn from the interior of the pelvis, the fragments follow from the traction communicated to them by the capsule. They are at the same time maintained in a position favorable for consolidation.

As I am aware of no case in which this dislocation has been reduced by the intra-abdominal route as proposed by Guibé, I cannot express an opinion.

Naturally, the after treatment by massage and early mobilization is indicated as in any case of dislocation.

## **CHOLELITHIASIS AND CHRONIC PANCREATITIS SIMULATING GASTRIC CARCINOMA \***

**By CURRAN POPE, M.D.,**

**AND**

**A. D. WILLMOTH, A.M., M.D.,**

**Louisville, Kentucky**

---

### **MEDICAL HISTORY AND LABORATORY FINDINGS BY DR. POPE.**

THE following case is reported under the impression that it may be interesting to the members of this society. Doubtless all of you will agree that a circle having a radius of about four inches from the ensiform cartilage as a center might be well termed "Hell's whole acre"; for more mischief may occur within that space, and it may offer more diagnostic problems, than almost any other portion of the body.

The patient, the subject of this report, is a female aged thirty-nine years, who gave the history of gastro-enteric disturbance. She has had numerous so-called "digestive attacks" which from a clinical standpoint suggested the possibility of gastric or duodenal ulcer. She has also had symptoms referable to the right lower abdominal quadrant indicative of chronic appendicitis. She represents a well-built, well-developed, sthenic type of woman. By the sthenic type, viewed from a radiological standpoint, is meant an individual who is short, thick and heavy from the ensiform cartilage to the pubic bone. She has lived an uneventful life, has been healthy and strong, is the mother of three children, and has had no other diseases of any practical importance.

With this clinical history she presented herself to Dr. A. D. Willmuth, and after a physical examination he referred her to me for clinical and laboratory investigation. The physical examination of this woman's abdomen showed quite a well-marked area of tenderness over the pylorus and also in the appendiceal region; there was no full-

---

\*Reported before the Louisville Society of Medicine.

ness or evidence of a neoplasm, nor did the physical examination reveal any apparent organic disease within the abdomen. There was no indication of syphilis and a previous Wassermann examination had been found negative.

In the laboratory investigation the urine was thoroughly examined, and with the exception of a decrease in the watery output (not over 700 c.c. in twenty-four hours), a marked decrease in the urea output, and the presence of substances indicating toxæmia from the intestine, there were practically no other abnormalities.

The fact is noteworthy that the only acid found in the stomach contents was a faintly acid (lactic) reaction to litmus. This probably was due to general acid factors and not to any acid digestion. The HCl, salivary digestion and solution showed good mastication and normal action of the salivary glands; whereas the quantity taken from the stomach showed mild stasis, but not of the atonic variety. Attention is called to the fact that the duodenal examination was quantitative, such an examination having been made possible through the courtesy of Dr. Max Einhorn, from whom I obtained the tubes necessary to make the analysis of the three ferments.

A thorough radiological examination was made. The habitus of the individual was normal. After giving an opaque meal the cesophagus was found normal, the meal passing through it rapidly; its position and peristalsis were correct. The stomach was hypotonic in character, and no filling defects were shown. It was of the fish-hook variety and lay vertically in the abdomen extending four inches below the umbilicus. It was normal in size and freely movable, possessed vigorous peristalsis of the four-cycle type, was incompletely evacuated after six hours, completely so after nine, showing evidence of mild gastric stasis.

The position of the pylorus was normal, it was freely movable, patent, and the opening was delayed; in fact, there was considerable well-marked pylorospasm present.

The duodenum was visualized, the position was normal, it was movable and patent. Peristalsis could be plainly noted, but the duodenal cap was quite irregular. In addition there was marked tenderness over the duodenum with a well-defined area of irregularity. It looked under the fluoroscope as though there might be a duodenal

ulcer, but its presence was questionable owing to the difficulty in examination of this woman, whose tissues were unusually heavy and resistant. The jejunum and ileum were negative. The ileocecal valve was competent, showing no trouble.

The appendix was low in its origin, partly retrocecal, was filled at the sixth to ninth hour, and emptied itself completely by the twenty-fourth hour. There was well-marked tenderness over the appendix, but in spite of this we decided it was negative.

The cæcum was normal, free, and was completely evacuated after the twenty-fourth hour. There was, however, an area of tenderness that coincided with the tip of the cæcum, or may possibly have been the right ovary.

The position of the colon was normal, it was movable, free, and patent; peristalsis seen; no antiperistalsis. Evacuated completely after twenty-four hours. There was a slight reduplication, however, at the hepatic flexure. At the twenty-fourth hour the entire meal lay within the pelvic colon and the rectum. We were inclined to believe the cæcal pain was the direct result of rectal spasm induced by an anal fissure which was present, and that the constipation from which this woman had suffered for a number of years was strictly of the anorectal type. In fact, this woman exhibited the typical twenty-four hour type of constipation. In such cases the food passes into the rectum and remains there sometimes for several days; or it may remain in the rectum for a while, then pass backward into the ascending colon, often into the transverse colon. I have observed this quite a number of times. It emphasizes the advantage of the opaque meal and röntgenology in the study of even rectal troubles. We can oftentimes locate the extent and character of the constipation, and in that way determine the particular medication necessary to overcome it. If we find reduplication and stasis at the flexures, then they should be treated in such way as will increase their capacity to empty themselves.

The rectum and pelvic colon were studied by the opaque enema, and it was noted that the pelvic colon did not rise freely from the pelvis with the fluid. It is just possible that we had to deal with a few adhesions at the pelvi-rectal junction.

When the second X-ray plate was taken we found a very marked irregularity in the upper side of the antrum right near the pylorus that looked extremely suspicious.

The radiologic examination of the lungs and heart was negative, and the abdomen showed nothing abnormal before the opaque meal was given.

In a résumé of this case, we find a history suggestive of many things, especially of gastric or duodenal ulcer, appendicitis, cholecystitis, or carcinoma. I neglected to state that the woman had lost seventeen pounds in weight which was at least suggestive of malignant disease.

We were sure from the results of our examinations that she was adynamic, asthenic if you please; that she was in what might be termed a "neurasthenoid" state; that she had anæmia; chronic pancreatitis; a well-marked functional disorder of the stomach; ano-rectal constipation, anal fissure, and that there was certainly some disease in the upper right abdominal quadrant. The question to be determined was, what have we to deal with?

The possibilities in this case resolve themselves into four categories: (1) incipient gastric carcinoma, (2) adhesions, pyloric and duodenal, (3) adhesions and cholezystic disease, (4) choleliths, cholecystitis and adhesions, the latter obstructing the cystic duct. Dr. Willmoth and myself in discussing the matter came to the tentative diagnosis of: "Surgical upper right quadrant of the abdomen," cholezystic disease, chronic pancreatitis, and possibly choleliths. We felt there was a strong suspicion of gastric carcinoma. Let us consider a few points in this case and see if we could not possibly have eliminated all these possibilities.

(1) *Carcinoma*.—We know that carcinoma is most frequent in the stomach. Lebert and Brunton say 25 per cent., Mayo 33 $\frac{1}{3}$  per cent., Rieche 49 $\frac{1}{2}$  per cent. It occurs most frequently in (a) the pylorus, and (b) the lesser curvature. We know the diagnosis is very difficult when the neoplasm exists at or near the pylorus. The stomach is inaccessible and difficult of palpation, especially in individuals of the sthenic type. In 33 $\frac{1}{3}$  per cent. of cases, according to most observers, there is no palpable tumor, and in 53 3-10 per cent., according to Mayo, there is no gastric motor insufficiency.

It is a strange fact that gastric carcinoma may pursue a latent course and exist undiscovered for a long time, and for that reason one should always be suspicious where the patient exhibits symptoms the least suggestive of carcinoma. Cachexia is always absent in the ear-

lier stages, and this woman was evidently in the early stage. Where carcinoma is engrafted upon ulcer the diagnosis is still further obscured, and this patient gave an indefinite history of gastric ulcer for many years. The presence of lactic acid in the stomach contents may be considered as proof of carcinoma in 80 per cent. of cases according to most authorities. However, this is not pathognomonic of carcinoma, as it may occur with diminished HCl and motor power. We had here, then, diminished motor activity, absence of HCl, the presence of lactic acid, and Boas-Oppler bacilli. However, these phenomena may occur in: (a) gall stones with pyloric adhesions, (b) in chronic gastritis, and (c) in carcinoma of the gall bladder. While an acidity may be frequently found in gastric carcinoma, it must not be forgotten that it is also not an infrequent phenomenon in benign gastric lesions. In nearly 80 per cent. of cases of gastric carcinoma HCl is absent, but the significance of this fact is difficult to prove in the absence of other signs of malignant disease.

That copious hemorrhage seldom occurs in gastric carcinoma, despite the tendency of malignant tissue to ulcerate, is explained by the limited blood supply to the tissues. The few existing blood-vessels are intimately associated with and are more or less compressed by the carcinomatous cells. While parenchymatous hemorrhage certainly occurs from the ulcerated surfaces, the blood exuded is insufficient in quantity to become microscopically evident. Constant bleeding may be easily proved, however, by chemical tests for occult blood in the faeces, and negative results after repeated examinations should be considered as evidence against the existence of carcinoma. On the other hand, frequent positive reactions on repeated fecal examinations constitute strong evidence in favor of carcinoma. Microscopic examination of the faeces for erythrocytes should be made to eliminate possible admixture of blood from the lower colon or rectum; in the latter event erythrocytes will invariably be found. The secondary anæmia commonly noted in advanced stages of the disease is probably due to constant hemorrhage from the neoplasm, together with the resulting malnutrition and toxæmia.

Of more decided diagnostic import is gastric pain which is present in the majority of cases at some time during the disease. No seral or gastric tests, such as the Salomon or glycyltryptophan reaction, can be considered as positively diagnostic.

The röntgenological findings are of the greatest importance in the diagnosis of gastric carcinoma. According to Carmen and Quimby of New York ninety-five per cent. of gastric carcinomata are discovered by the X-ray. Among the most notable röntgenological manifestations are filling defects occasioned by projection of the neoplasm into the lumen of the stomach. Hour-glass deformity occurs in involvement of the pars media and gastrica, giving a less sharply defined contour than in hour-glass due to ulcer. Filling defects may occur in other lesions, but their differentiation should not cause serious confusion. Pyloric function may be altered by gaping or obstruction caused by infiltration and consequent stiffening of the muscular ring, interfering with sphincteric contraction. Obstruction is indicated by six-hour or longer barium residue.

The question of *syphilis* should be considered in obscure gastric lesions, as there is always a possibility of luetic involvement. It may occur either as a gumma, or as a diffuse infiltration of the stomach wall. While the clinical picture is not unlike that of non-specific disease, the results from therapy are decidedly different. Eusterman gives a symptom-complex as probably indicating gastric syphilis "in the combination of anacidity, subacidity, or achylia, with a syndrome resembling benign ulcer in some respects and gastric carcinoma in others, and accompanied by röntgenological findings suggestive of carcinoma." Gastric syphilis closely resembles malignant disease of the stomach in its various phases. Achlorhydria is characteristic of the disease; an ulcerating gumma will give a positive occult blood reaction in the faeces; healing of the ulcer may result in cicatrization with contraction stenosis accompanied by evidence of stagnation characteristic of pyloric carcinoma. It becomes evident, therefore, that little reliance can be placed upon the history or the clinical features in differentiating the two lesions, and the deciding factor must be serologic examination.

The diagnosis of gastric carcinoma, despite the multiplicity of modern methods of scientific investigation, is a problem of the greatest magnitude, particularly in connection with early cases. The insidious development of the disease is often the cause of diagnostic failure. As the most favorable therapeutic results are to be obtained only in the early stage of the disease, surgical exploration must be included among the diagnostic methods in doubtful cases.

The argument can be adduced in this case that it was far better to resolve doubt in a questionable growth at the accessible pylorus, than to incur the risk of delay. That was our conclusion with regard to the question of carcinoma.

(2) *Pyloric and Duodenal Adhesions*.—This woman suffered from pain and tenderness with mild gastric delay. It must be remembered that the pylorus may be mobile or fixed, depending upon the degree of the adhesions, and there was distortion upon the plate of both the pylorus and the duodenum. There was no ulcer present for the reason that the adhesions were no more prominent at the beginning than at the close of the examination. These are the most interesting points in regard to pyloric and duodenal adhesions.

(3) *Adhesions and Cholecystic Disease*.—We had the possible presence of adhesions at the duodenum and at the pylorus. The duodenal bucket showed a slightly opaque bile which on analysis gave evidence of chronic pancreatitis, and we believed there were adhesions involving the cholecyst. If choleliths were present we believed they obstructed completely or nearly completely the cystic duct. The hepatic duct was discharging freely, and slightly opaque bile showed a mild degree of cholangitis.

(4) *Choleliths, Cholecystitis and Adhesions*.—Taking into consideration all the facts embraced under heading No. 3, we were reasonably certain there was a cholecystitis from the appearance of the bile, and in the absence of gastric carcinoma, that we had to deal with adhesions of the pylorus, duodenum and cholecyst, with the probable presence of choleliths.

Thus the diagnosis of "surgical upper abdomen" was made, and the opinion given that surgical work under this diagnosis became at once needful. The case then became a surgical one and passed automatically from the hands of the internist.

#### OPERATION AND PATHOLOGICAL FINDINGS BY DR. WILLMOTH.

The case reported was of unusual interest to me from more than one standpoint. The patient was the wife of a physician, which naturally made the burden a little heavier than in the average case. After making a careful physical examination in my office, I thought the patient most likely had a duodenal ulcer. She was seventeen pounds short of her usual weight, and this reduction had occurred during the

last four months. She gave such an indefinite clinical history as to cause me to suspect gastric ulcer.

I thought the woman was somewhat sallow in color, although it was a dark day and the artificial light made this questionable. She thought her color was due to sunburn more than any other cause. After a general examination the patient was referred to Dr. Pope for further clinical and laboratory investigation.

The pelvis was negative so far as could be determined from my examination of her pelvic organs. I asked particularly regarding her secretory and excretory functions, and she said there was nothing abnormal. My examination revealed nothing to account for her loss of weight or any of the gastric or other disturbances from which she complained.

After Dr. Pope concluded his investigations and we had a chance to consider the data thus obtained, this woman presented almost perfectly the symptomatic tripod of malignancy, that is the presence of Boas-Oppler bacilli, the presence of lactic acid, the absence of HCl. These facts in connection with her loss of weight made us strongly suspect gastric malignancy possibly engrafted upon an ulcer. This information we transmitted to her husband. We did not tell the woman the whole story because we thought it wise not to do so.

The presence of choleliths was not shown by the X-ray plate. There was nothing we could interpret as indicative of biliary calculi, nor to strongly suggest their presence. There was no history of repeated attacks of colic, although she had complained for a number of years of so-called "dyspepsia"; in other words, she had more or less gastric disturbance from time to time which she described as "indigestion."

With the history outlined we believed that we were possibly dealing with beginning gastric malignancy, sufficiently early to permit of its removal, and the patient was subjected to celiotomy. It was our intention if the disease was recent and not too extensive that the involved portion of the stomach would be removed. Her husband who was present at the operation fully understood the situation.

When the abdomen was opened through a right rectus incision, it was readily determined that there was practically no involvement of the appendix. Although there was some irregularity and evident

pathology in that region, there was nothing we could say was referable to the appendix itself.

The feature in which we were most interested was the possibility of gastric carcinoma, and my fingers were quickly passed upward to the pyloric region to determine whether she had beginning malignancy. I could feel nothing to indicate that there was even an ulcer in that region. No induration or nodulation could be felt about the pylorus or stomach, but to determine this with absolute certainty the incision was extended upward until the stomach could be brought into view, and in a few minutes we were able to state positively that the woman had neither a gastric carcinoma nor an ulcer. I even incised the lesser omentum and inserted my fingers into the lesser cavity to palpate the posterior gastric wall. This examination was also negative. However, we had already noted signs pointing in the direction where the greatest pathology existed. The pyloric end of the stomach and the duodenum were found adherent to the cholecyst, and the gastrohepatic omentum was also fixed by adhesions.

After thoroughly examining the viscera it was readily determined that the enlarged cholecyst was densely adherent and palpation revealed a number of choleliths. After separating the adhesions the large and indurated pancreas was easily demonstrated.

The appendix was removed and the cholecyst incised and four choleliths were extracted. There was no obstruction of the cystic duct nor at the ampulla of Vater. After determining the patency of the duct I decided against cholecystectomy for two reasons: First, it was not the so-called "strawberry gall bladder" which would most likely have terminated in malignancy, and second, the cholecyst was left for the purpose of facilitating drainage. Of course, drains can be placed in the common and the hepatic ducts, but this incurs greater risk to the patient. It was decided to leave the cholecyst for the purpose of drainage to relieve the existing chronic pancreatitis. There was no duodenal obstruction or ulcer, and the jejunum was examined and no ulcer found there. There was no obstruction to the biliary apparatus and a large tube was placed in the cholecyst for prolonged drainage.

The case is interesting because all the symptoms were suggestive of malignancy, yet they were entirely non-malignant in character, and we are made to think of many other manifestations which may be induced by appendicitis, or cholecytic disease. William J. Mayo told

me recently that he had seen three distinct gastric hemorrhages originating reflexly from appendicitis, which led him to a further study of symptoms referable to the stomach which might be reflex in character. What percentage of cases present gastric manifestations I am unable to state, but a great many authors mention this fact. Kemp in his recent work on diseases of the stomach states in from seventeen to twenty-three per cent. of cases reflex causes were noted which interfered with the chemical composition of the stomach contents and gave rise to symptoms similar to those presented in the case here reported. This proved to be one of the cases coming within the small percentage where the symptoms strongly indicating malignancy were really produced by an irritated cholecyst and mechanical factors, as well as by reflex disturbances.

The woman has made an uninterrupted recovery, and is eating and sleeping well. The tube has not been removed, and drainage will be continued just as long as it is possible. She has been told why the tube should remain in position six months or longer if possible. This continuous drainage is for the purpose of relieving the chronic pancreatitis, the result of irritation from the cholezystic disease, adhesions, mechanical and chemical factors, etc.

A few words may be permitted on the question of chronic pancreatitis. Many of those who have written on the subject speak of pancreatitis as being due to infection. I believe, however, the consensus of opinion to-day is that chronic pancreatitis, such as was present in this case, is not the result of infection but of chemical irritation from the bile, and that prolonged drainage of the cholecyst and its ducts will relieve the larger percentage of these cases. There are very few which will not be relieved by continuous drainage either directly through the cholecyst, or by means of a "T" tube where cholecystectomy is performed.

This case fell within that number which required exploratory incision to determine the existing pathology. As has been well said by Deaver, "we learn things in two places, (a) at the operation, and (b) at the mortuary slab!" In many instances the laboratory findings merely aid us in the way of making certain preparations. Such findings are not always absolutely pathognomonic, as in this case, and we finally have to resort to exploratory incision to determine the real nature of the pathology.

We advised this woman to undergo immediate operation for the reason that we believed malignancy might be present, and the earlier the operation was undertaken the more likely would we be able to resect the portion of the stomach involved; that by doing a posterior gastro-jejunostomy we might be able to relieve her of the malignancy and prolong her life for many years. Fortunately for the patient such an operation did not have to be performed.

Before closing I desire to again take issue with those who argue against continuous drainage of the biliary tract in the attempted relief of chronic pancreatitis. The reason the pancreas becomes involved in cholelithic disease is because of the back pressure. The real function of the cholecyst is to relieve this back pressure. The presence of a sphincter at the ampulla of Vater is well known; it was described and brought prominently before the profession several years ago by Ardis and one or two others. As a result of this sphincter there is back pressure on the outlet of the hepatic duct into the cholecyst, and indirectly through the ducts of Wirsung and Santorini into the pancreas. It is in this way that chemical agents are forced into the pancreas which originate the pancreatitis. If one expects to relieve the pancreatitis the quickest way is to overcome or prevent the back pressure. The pressure at the sphincter cannot be relieved at once by removal of the cholecyst, because it takes nature a long time to produce sufficient dilatation of the common duct to accomplish this. The quickest way is to drain through the cholecyst, or if cholecystectomy is performed, to drain through the cystic duct.

Three or four years ago I had the pleasure of seeing Dr. Deaver, at the German Hospital, Philadelphia, Penna., use a "T" tube which impressed me favorably where prolonged drainage became necessary after cholecystectomy. It caused me to study further the question of persistent drainage in such cases. In the October, 1917, issue of *Surgery, Gynaecology and Obstetrics*, is an article by Deaver giving his experience in about twelve hundred cases where the cholecyst was drained, and in which he reports that a number of his patients have worn this "T" tube as long as three years. He further states that in over four per cent. of the cases of cholelithic disease the patients had been previously operated upon and not drained without permanent relief. In some instances choleliths were overlooked at the original operation, and in others the pathology was such that it could be relieved

only by continuous drainage. When drainage was discontinued for a time the symptoms reappeared; in other words, so long as drainage was continued the patient was perfectly comfortable, but when discontinued the symptoms recurred. He reports two or three instances where the patients refused to have the tubes removed, stating that was the only way they could obtain relief.

Continuous drainage is certainly preferable to abdominal pain and everlasting discomfort. It is the quickest way to obtain relief from chronic pancreatitis; and while no one can state positively that the patient will remain permanently well, this method of drainage is through normal channels and recurrence of pancreatitis has not been reported where prolonged drainage was practiced.

In the case reported the concretions were in the cholecyst, the common duct was unobstructed. If the common duct had not been patent no bile could have reached the duodenum, and the patient would have had profound cholangitis. Even though the common duct was unobstructed the patient developed pancreatitis, and the most rational procedure was to drain through the cholecyst. Had cholecystectomy been performed I would have instituted drainage by means of the "T" tube, placing one end in the common duct, the other in the hepatic duct, the main stem of the tube emerging through the abdominal wall. Regardless of the contrary views of others, I have been forced to the conclusion by personal experience that prolonged drainage is indicated in cases such as the one herein reported.

#### CLOSING REMARKS BY DR. POPE

I believe the proper method of investigating a case is: (1) to make a careful clinical study, (2) a thorough physical examination, (3) to employ every form of laboratory test that can possibly assist in completing the diagnosis.

There seems nothing more ridiculous than for the clinician to expect the laboratory worker to make an accurate diagnosis based upon a single sample of tissue or fluid, as happens almost constantly. For instance, a single specimen of urine—taken at any hour during the day without reference to the total quantity voided during twenty-four hours, or any other information in relation to the individual patient—is sent to the laboratory with the expectation of learning something definite from it. Of course one may learn something, but not all that

is possible to be known; and the more I see of laboratory methods, the more I check the work of others along this line, the more do I come to the conclusion that there is just as much necessary technique in this as in the most complicated surgical work. There is only one way to do laboratory work, and that is the right way.

If you want to examine a specimen of urine to make a diagnosis, get a twenty-four-hour specimen; and if you want to get all the light possible upon an obscure case you must have recourse not only to your clinical acumen and all the evidence obtainable by physical examination, but the information thus obtained must be supplemented by all laboratory measures which have been discovered for the betterment of this analytical work.

As to the question of diagnosis during the period of delay: This is the factor which impresses us perhaps the most forcibly, and is one of the most difficult that we encounter. The problems are many and the possibilities of error are great. To anyone who studies the intimate and interesting rhythm from above downward in the gastro-enteric tract, the interlocking influence of intestinal activity and hormone stimulation, the reflex influences which may travel from below upward (from the anal aperture to the cardiac end of the stomach)—when we think of all these factors and realize that each and every one of them may be modified many times and by many conditions, it seems to me that the only sensible, sane and scientific attitude is to insist upon more light, that upon this depends the entire crux of the situation. We cannot acquire too much light nor scientific knowledge concerning our cases if we really want to make accurate diagnoses as possible and to eliminate the possible errors which are eliminable. Then we have reduced our case to certain categories, and when this has been done we can at least with some satisfaction to ourselves and some reasonable comfort to the patient offer advice that is useful and in many instances correct.

In this particular case I take it that we made no mistake; that is the essential crux of the situation. We made the diagnosis correctly of a surgical lesion within the upper abdominal quadrant, one that amply justified celiotomy. We formulated exactly, before the abdomen was opened, every possibility that could occur, and behind that formulation was a good reason for each and every one of them. So I take it we did not in any sense of the word overlook a single point or

make a single mistake, because we had foreseen and anticipated every possibility. And I think that is not only one of the most interesting and fascinating features of the work, but it has an influence upon the physician himself that is far-reaching and beneficial. It tends to make him work hard, that is to think—and thinking is hard work. And if we can get our facts and figures to such a point that we will really think and reason about our cases, the chances are that we will eliminate many errors that are eliminable. In fact, my observation has been that for the most part our serious mistakes in diagnosis are based more upon carelessness or thoughtlessness of the physician than upon crass or gross ignorance. Many times have I said to myself that had I weighed more carefully all the facts that I had or knew, I would not have made a mistake. I believe it is only by carefully controlling ourselves, "holding our own nose to the grindstone," that we can hope to overcome the many errors attributable to the frailty of human nature.

## WHAT SURGERY OWES TO MILITARY SURGERY: A GREAT PIONEER IN CLINICAL SURGERY\*

By JAMES J. WALSH, M.D., PH.D.

Medical Director of The Sociological Department of Fordham University and  
Professor of Physiological Psychology at the Cathedral College, New York  
City.

AT the present time when so much of the attention of the medical profession is, if not necessarily at least patriotically, occupied with matters relating to the war it is extremely interesting to turn to the pages of the early history of modern surgery in Europe and note what magnificent contributions were made to surgery by the military surgeons. I deliberately choose the epithet Modern Europe and yet the period that I am about to refer to as a really great pioneer epoch in modern surgery is that of the later Middle Ages, and especially the thirteenth century. Most of us would probably not associate the idea of a great advance in surgery having come as the result of the military experiences of the European surgeons who accompanied the troops that went on the later Crusades, and yet as a matter of fact we now realize that they brought back with them a magnificent accumulation of practical and scientific information in surgical matters which was very soon made available for their colleagues in civil life at home.

There is to a very great extent the inclination to presume that the men of this period neither had the time nor the inclination, nor above all the incentive and intellectual power, and beyond all they are supposed to have lacked the surgical sense to be able to take advantage of their clinical opportunities, no matter how ample these might be. This is, however, as we have come to realize very clearly in recent years, the merest of presumptions, which has been completely upset by the study of original sources in the history of surgery during the past generation.

---

\* This paper is an extension of one part of a discussion with the presentation of old text books before the Section on The History of Medicine of the New York Academy of Medicine on "What Surgery Owes to the Military Surgeons of Old Times."

All history has been revolutionized during the past century by the reversion once more to the original documents. In this great development of history, which has come as a consequence of research and investigation into original sources, nothing has been more astounding than the discovery that our modern surgery, of which we are deservedly so proud, is not only not new, but is really very old. Our modern phase of surgery is only a repetition of a preceding phase of surgical history which developed in the later Middle Ages, when a group of wonderful original surgical workers in France and Italy, most of them professors at the Italian and French universities of the time, succeeded in making what would have seemed to our immediate forefathers in surgery quite incredible developments in their favorite specialty. As a thoroughgoing demonstration of this I need only mention the fact here that these old-time surgeons of the later Middle Ages discussed most of our modern operations, at least in general, described with a good deal of elaboration the technic of both diagnosis and treatment for surgical conditions and laid down the principles on which surgery in theory as well as in the practice of particular operations could be successful. When this information began to come to us, at first it seemed utterly beyond belief. The details amply justify what has been said, however. These old surgeons described operations, for instance, which even required opening of the skull for tumor and for abscess as well as for fractures, suggested opening of the thorax for pus and for other fluids, and went into some detail with regard to a number of operations within the abdomen. They insisted, for instance, that patients with wounds of the intestines would surely die unless these were repaired, described how these operations should be done and invented various instruments and modes of treatment for their successful carrying out. They did operations for the radical cure of hernia, placing the patient in an exaggerated Trendelenberg position, on a board reclining against a wall, in order that the intestines might drop away from the site of operation, and in many other ways showed that they were facing the surgical problems of all times not only with intelligence and ingenuity, but with real genius for the solution of the surgeon's most difficult questions.

Of course they could not have done such extensive operations without an anæsthetic, but it is now perfectly certain that, for some two

centuries, at least, all the important operations in surgery in Europe were done under an anæsthetic. We know what the anæsthetic was and know that it would be useful, though neither as certain nor as safe as ours. Such extensive operating must surely have been followed by septic conditions and an awful death rate, only that these old-time surgeons had made some discoveries in antisepsis as well as in anæsthesia. They used strong wine as an antiseptic, boasted of getting union by first intention, insisted that a surgeon who got pus in a wound had made a technical mistake in his work and talked proudly of their very pretty linear cicatrices, which could scarcely be noticed only a short time after the operation.

We know these items of surgical history not by tradition, nor even by the description, however detailed, at second hand from historians who might be enthusiastic about a favorite period, nor from biographers who might be exaggerating the significance of what was accomplished by their particular subject, but from the actual textbooks in surgery of the olden time. These have been republished in recent years, they were all of them originally printed in the Renaissance time within a half century of the invention of printing and the early printers conferred an immense obligation on the medical profession and perhaps never did anything better for history than their venturing to print magnificent editions of these old surgeons.

Practically all of this wonderful knowledge of surgery was gained as the result of experience in the wars of the mediæval period and particularly the Crusades. Now that we, in this country, are engaged in such another great migration of armies in another Holy Cause it may be interesting to review what was thus accomplished, in the hope that, in our time at least, war may be as beneficent in the contributions that it affords to medicine and surgery. For medicine benefited greatly too, though we are not so much concerned with that here. It was these surgeons of Italian and French universities who in the thirteenth and fourteenth centuries established for all time the status of mercury in medicine. The use of mercury is probably the greatest therapeutic triumph that was ever achieved, and we owe it to them. They brought back from the East a number of drugs that were of value, but above all they taught the value of water and air and exercise for ailing

people. If nothing else had been accomplished, these would have represented very great advances in medicine.

It would be useless to try to tell the story of a whole group of these surgeons, because it would take a book rather than an article. Besides, there would be inevitable repetitions. I am going to take a single one of them, who happens to represent the culmination of a surgical dynasty as it were, a group of men who were masters in surgery if any ever was, and this one took the time to write out the experience of his father and brothers as well as to cite the classical surgical text-books that had preceded them, so that he represents the veritable climax of a really great era in surgery. His name was Theodoric. Strange as it may seem to modern times, he was a bishop as well as a surgeon, and his being a bishop instead of being an obstacle to his work in surgery seems rather to have been a help, for he was enabled to devote himself to his writing without the hindrance of exclusively professional work. Though he practiced surgery freely and, indeed, according to well established traditions, made considerable money out of his profession, leaving large sums to be distributed after death to charity, especially to hospitals, his official position afforded him the leisure and the dignity that enabled him to bring surgery properly before the world of his time.

Lest it should be thought that my own fondness (and that word fond always has a little innuendo, at least of foolishness, about it) for the Middle Ages and my recognized penchant for bringing out all the significance of the work done by the Churchmen of the mediæval period might possibly lead me to exaggerate the significance of Theodoric's work, let me quote the latest writer on the subject here in America, Dr. Albert H. Buck, whose volume, "The Growth of Medicine from the Earliest Times to About 1800," has done so much to give us a vivid picture of the medicine of the past.<sup>1</sup>

Dr. Buck said of the subject of our sketch: "Theodoric of Lucca, known as Bishop Theodoric, was born 1206 A. D. While still quite a young man he joined the recently established order of preachers, and not long afterward was appointed Almoner (Poenitentarius) to Pope Innocent IV. Eventually he became Bishop of Cervia, near Ravenna. By special permission of the Pope, he was able to complete

---

<sup>1</sup> *Yale University Press, New Haven, 1917.*

the surgical training which he had received from his father, Hugo of Lucca; and thus, while he still held the office of Bishop, he practiced surgery to some extent in Bologna. In course of time his practice became very extensive and also very lucrative; as a result of which he was able to leave a large fortune to various charitable institutions. The first printed edition of his work in surgery appeared in Venice in 1498, and was followed by numerous later issues.

“‘Theodoric,’ says Neuburger, ‘was a most uncompromising advocate of the dry method of treating wounds.’ His words are these: ‘For it is not necessary—as Roger and Roland have said, as most of their disciples teach, and as almost all modern surgeons practice—to favor the generation of pus in wounds. The doctrine is a very great error. To follow such teachings is simply to put an obstacle in the way of nature’s efforts, to prolong the diseased action, and to prohibit the agglutination and final consolidation of the wound.’

“In his enumeration of the different means that may be employed for arresting hemorrhage, Theodoric mentions cauterization, tamponading, the application of a ligature, and the complete division of the injured blood-vessel. He attached great importance to the proper feeding of the patient. In Book III, Chapter 49, of his treatise on surgery, he gives minute instructions with regard to the proper manner of employing a salve made with quicksilver, and at the same time he mentions the fact that he observed a flow of saliva as one of the results of its use.

“The expressions ‘healing by first intention’ and ‘healing by second intention’ are encountered for the first time in the writings of Brunus (or Bruno), a surgeon who practiced in the cities of Verona and Padua about the middle of the thirteenth century, and who was a vigorous advocate of the dry method of treating wounds.”

It is worth while going to Gurlt’s great history of surgery<sup>2</sup> to get the rest of Theodoric’s history. Gurlt has some ten pages with regard to him, most of it in rather small type, so that altogether he has some six or seven thousand words with regard to him. Gurlt went for his information direct to the *Cyurgia edita et compilata a domino fratre Theodorico episcopo Cerviense*—“Surgery Edited and Compiled by Lord Brother Theodoric, Bishop of Cervia.” This

---

<sup>2</sup>“Geschichte der Chirurgie Und Ihrer Ausübung von,” Dr. E. Gurlt, Band 3, Berlin, 1898.

volume was published in 1498 in the Venetian Surgical Collection (*Collectio Chirurgica Veneta*). It is no end of surprise to find what this book, printed within ten years of the discovery of America, from a manuscript publication that had been written about two centuries and a half before, and circulated freely in the academic world, at least, contains of modern surgical principles and practice, and how marvelously the old Bishop, whose father had been a surgeon and two if not three of his brothers had been surgeons, had gathered together from observation and from the family note books as to the eternal actualities of clinical surgery.

Theodoric, whose family name was Borgognoni, was the son of Hugo Borgognoni, known as Hugo or Hugh of Lucca, or sometimes as Ugo da Lucca, or, by the Latinized form *Ugo Lucanus*, because he was born in the little town of Lucca. This Hugo was manifestly a very great surgeon. We hear of him first as a man who had made a reputation for marvelously successful surgical practice in Lucca and who was called to serve as city physician and military surgeon to their troops by the municipal authorities of Bologna. In 1218 he accompanied the Bolognese troops on the Crusade and was present at the siege of Damietta and seems to have spent some three to five years in active surgical military service. When he returned to Bologna he was given the position of Municipal Medico-legal Expert, or physician to the courts, which evidently included some jurisdiction over sanitary matters in Bologna. The municipal statutes under which he was selected for this position are among the oldest monuments of legal medicine in the Middle Ages.

The distinguished surgeon, in spite of the hardest kind of work, and the most serious responsibilities, and the long series of campaigns, including the campaign with the Crusaders in the East, undertaken when he was already approaching sixty years of age, lived to be nearly a centenarian. He died just after the middle of the thirteenth century; we are not quite sure when. He was alive in 1252; he was surely dead in 1258.

Of his five sons, three, at least, became physicians, and, under their father's tutelage, learned the secrets of his surgical practice and of his observations with regard to medical matters generally. I say deliberately the "secrets" because medical and surgical knowledge

was at this time, in accordance with the old tradition in Asclepiad families, still passed on from father to sons under the seal of secrecy. This seal was to be broken by the greatest of his sons, Theodoric, who was apparently very proud to tell this story of how that wonderful man, Master Hugo, had cured nearly all kinds of wounds "with wine and flax and a properly arranged dressing which he knew how to make." He not only cured them, but "he made them heal solidly as before and succeeded in obtaining very beautiful cicatrices without any ointment."

Theodoric, following the heredity of longevity in the family, lived also well beyond the age of ninety, so that his life covers nearly the whole of the thirteenth century. He was born in 1205, he died January 9, 1298. As a young man of twenty-two or twenty-three he entered the recently established order of preachers, which is still not only in existence but flourishes here in distant America seven centuries later. It was founded by St. Dominic, and we know it as the Dominicans. In spite of his entrance into the cloister, this devotion to the religious life brought, as Gurlt notes distinctly, no hindrance to the completion of his education in medicine, and indeed special arrangements seem to have been made so as to enable him to use his surgical knowledge to the best possible advantage for the benefit of the ailing.

He became the Chaplain of the Bishop of Valencia, his Bishop manifestly granting him permission to continue his surgical studies and work, and then later he became a Penitentiarus, that is a confessor with special privileges for dealing with unusual cases appointed by Pope Innocent IV. Theodoric was evidently in great favor with the ecclesiastical authorities, for a little later he was made Bishop of Bitonti in the Province of Bari, and, when he was about sixty years of age, transferred to the Bishopric of Cervia, a little town not far from Ravenna.

Theodoric's treatise on surgery contains four parts or books. The first book treats of wounds, ulcers, hemorrhages, fatal injuries, wounds of nerves, inflammations and tetanus. The second book concerns the same subjects, but with special relation to the separate parts of the body, from the head to the feet. In the third book fistulae, cancers, warts, other forms of tumors and other subjects having rela-

tions to overgrowths of various kinds are treated. The fourth book is concerned with headache, the diseases of the eyes, paralysis, epilepsy, gout and other conditions that disturb joints or bring about deformities which may be the subject of surgical intervention.

Not a little of this first book follows very closely Bruno of Longoburg, usually known simply as Bruno or Brunus, who had written on surgery shortly before. Indeed, there has been some question in recent years as to who it was that followed the other, for we know nothing at all of Bruno's life, and have only his book. Theodoric does not mention him, but, then, he seldom mentions any of the recent contemporaries, though he does mention the older authorities of surgery, and not infrequently disagrees with them on certain points. This failure to give due credit to Bruno has sometimes been counted as a fault in Theodoric, but he was following the custom of the time. After all, it would be extremely difficult for a writer on surgery in our time to refer faithfully everything that he incorporated in his book from others to their original authors, or even to those from whom he obtained them. There is a body of tradition in surgery from which everyone draws and there are very similar modes of expression. Textbooks on medicine and surgery in our time follow one another very closely in certain places, so much so as to make it clear that there has been special influence exerted and yet personal credit is not always given. Sometimes this would seem to be plagiarism, but as Mark Twain once said, "I am not sure from whom I got that good thing, but I am quite sure that whoever it was he got it from someone else."

#### SURGICAL PRINCIPLES APPLIED

It might very well be thought that this good bishop-surgeon of the thirteenth century laid down very wisely some of the general principles of surgery, which fortunately, though perhaps more by a happy accident than otherwise, proved to be along the lines which long subsequent scientific surgical progress came to recognize as fundamental, but it would perhaps readily be imagined that as regards the practice of surgery he was woefully lacking in knowledge of details. As a matter of fact only a little study of his work on surgery is needed to prove almost astounding in its revelation of detailed knowledge of the practice of surgery as outlined by him. When he takes up the

various parts of the body he gives intimate details of technic in diagnosis and treatment that are almost incredible in their anticipations of modern knowledge. Surgery had sunk to so low an ebb in the century preceding our own that it seems almost impossible to credit that there could have been such excellent knowledge of surgery seven centuries ago. Bishop Theodoric, however, coming at the end of a line of thoughtful surgeons, had accumulated an immense amount of valuable information which he proceeded to set forth for posterity and made available not only for his own generation in Italy but for those of centuries later everywhere throughout the surgical world.

In the general treatment of wounds Theodoric insisted that the most important procedure was the cleansing of the wound and after that the careful bringing together of the wounded edges either by deep or superficial sutures, more or less of the flesh being included in them. He refused to follow the custom of those, such as Roger and Roland, his great predecessors in the teaching of surgery, who declared for the use of a wick of absorbent material for drainage. Theodoric thought that no error could be greater than this for it would surely impede nature, prolong the disease, prevent the union and consolidation of the wound by first intention, encourage uncleanness in the part and sadly hamper the process of repair and cicatrization. In wounds accompanied by loss of tissue substance warm wine should be used freely and dressings placed directly over the wound to encourage regeneration and then the parts should be drawn together from all sides so that the portion necessary for repair should leave as small an amount of skin surface lacking as possible. He agreed with Galen and other antique authorities in medicine that it was impossible to restore the skin, but underlying tissues could be encouraged to repair.

In the treatment of hemorrhage there occurs his description of the use of the ligature and of the technic of securing bleeding vessels. He believed firmly in the benefit to be derived from local pressure and the use of tampons and, as Gurlt remarks, describes exactly how to use them according to the latest modern practice. Whenever a pulsatile vein, a blood-vessel which he also calls an artery in other places, bleeds freely the vessel should be cut across if it is only wounded on the side so as to enable it to contract properly and also to prevent the occur-

rence of an aneurism which he calls an *emborisma* in the wounded part. He says I have often brought people to recovery who had a wounded pulsatile artery without the subsequent occurrence of emborisma. Where contraction of the artery, that is of its severed ends, followed by pressure did not bring about cessation of the hemorrhage a ligature should be applied directly to both parts of the cut artery. Evidently, however, he believed a great deal in direct finger pressure in bringing about the cessation of hemorrhage. He warns against the use of the cautery or cauterizing materials for this purpose unless under special circumstances. When they are used they should be strongly cauterizing materials or styptics like vitriol or quick lime.

Theodoric was a firm believer in good nutrition as the best possible basis for successful surgery. He says, "Since, therefore, nature herself cannot bring about the manufacture of good blood without proper nutriment nothing avails more in the healing of small as well as great wounds as the care of the nutrition of the patient. The physician must above all not be ignorant of the kind of food materials that manufracture [generate is the word he uses in Latin] good chyme and good blood. Out of such materials the wounded man should be fed in order that a suitable diet should bring about a restoration to health and the renascence of the flesh and the restoration of the continuity of the wound." He enumerates the number of foods and drinks as also some potions and mixtures which are especially suitable for wounded patients to give them back their health and strength as soon as possible.

With regard to compound fractures (*fractura cum vulnere carnis*—fracture with wound of the flesh) Theodoric is manifestly prepared to insist on great care as to manipulations. He suggests that the bandaging should be rather simple and that a compress should be placed over the wound moistened in warm wine. This should not be touched for ten days and then the whole limb should be washed off with warm wine. In ordinary fractures the limb should be rubbed with lard and then afterwards with honey and a stiff bandage that had been soaked in eight beaten eggs should be wrapped around it to help to maintain the position. He warns that the albumen bandage must not be used in compound fractures and the lard and honey salve must not be used. After reposition in an ordinary fracture

the bandage should not be removed for some two or three weeks. Compound fractures should be seen oftener but not fussily. He emphasizes the necessity for particularly good food in cases of fracture of the bone in order to bring about proper union.

#### SKULL AND BRAIN SURGERY

Fractures of the skull and other injuries of the head receive a good deal of attention from this very practical old surgeon. Wounds of the scalp and soft parts of the head generally if uncomplicated should be treated by placing a compress of linen that had been soaked in good hot wine over the wound, any hair in the neighborhood having been shaved. Care should be taken to bring the edges of the wound as carefully together as possible. Theodoric thought it a mistake to follow Avicenna in this matter and put sutures in the scalp, and very probably he had often seen serious results from infection spreading beneath the scalp because of this practice. Evidently just as little manipulation as possible was the safest for the patient, and above all Theodoric tells how strenuously his father, Hugo, warned against the misuse of salves in these cases. The recent use of small pieces of adhesive plaster instead of stitches in bringing such wounded edges together would seem to be a reversion to Theodoric's practice, though he would probably have objected to the plaster and employed simpler means of bringing about co-ap-tation.

Injuries of the skull itself, contusions and fractures are treated at some length. He suggests that there may be fractures of the skull which are really only linear cracks and which may readily escape notice unless special care is taken to look for them. These capillary fractures (*fractura capillaris*) may be discovered easily and certainly, however, if some black fluid is poured over the area when the crack stands out. All these mediæval surgeons have something to say with regard to fracture by *contre coup*, that is fracture not at the part of the skull where the violence was inflicted but on the opposite side. Whenever not only the skull is fractured but the coverings of the brain are also injured Theodoric warns that the prognosis is very serious. A good many of such cases, however, recover and an absolutely fatal prognosis must not be given until the course of the case has been watched for a while.

Not only may fractures of the skull with injury to the membranes of the brain not prove fatal but it may even happen that a certain quantity of brain substance itself may be lost without an inevitable fatal termination. Theodoric says that he had seen a number of such cases or as he puts it "with regard to this large experience makes it quite certain. I have seen many patients recover completely though they had suffered from injuries to both the membranes of the brain and a few even recover from whom not a small quantity of brain substance itself had been lost." Manifestly there had been some question raised as to the possibility of such a severe injury not proving fatal and so Theodoric adds emphatically, "Of this we are as sure as we are of death."

He had even seen a case with a very extensive loss of brain substance in his father's practice in which complete recovery took place. He says, "I knew a man from whom one of the cells of his brain [probably a ventricle, as Gurlt suggests] had been completely evacuated and yet in spite of this he recovered perfectly." The surprise for both himself and his father was that the man's memory was not affected and he was well able to go on with his trade, that of a maker of benches, quite as he did before. Evidently Theodoric and Hugo had had some such almost astounding experience as our own famous "crowbar" in which a premature blast blew a tamping iron through the skull of a quarryman, carrying away a great deal of the brain substance from one hemisphere, and yet the man recovered completely.

When fracture of the skull is present, depressed portions should be removed gently, if possible, but *must* be lifted if the patient is to recover. When the depressed skull is so wedged as to prevent removal in any other way a series of openings should be made with a *perforatorium* or *trapanum*, a trephine, and then with instruments which are called *levatoria*, elevators, pieces of bone should be lifted out and the roughness of the edges of the bone around the opening smoothed off so as to avoid further injury to the brain. The removal of pieces of bone is greatly facilitated, just as soon as an opening in the skull has been made, by introducing a special iron lever with a lenticular or lens-like terminal beneath the skull and then with a wooden mallet breaking off wedged parts. This can be done without in any way injuring the coverings of the brain. After this the wound

should be dried and freed from all blood with cotton and sponge and above all the dura mater should be touched only with the most delicate old linen wet with warm wine, and a dressing soaked in this should be placed over the opening and the head thoroughly covered.

#### SPINAL SURGERY

In injuries of the cervical vertebræ the prognosis is stated to be very serious, but something with regard to it can be determined by observing certain symptoms. Theodoric says: "If you wish to know whether the patient will get better or not consider whether his hands are relaxed and without feeling or control, as if dead. Whether the patient can move them or not, whether he is capable of extending them, whether he feels it when they are pinched or pricked with a pointed instrument. If all these are negative the condition is mortal. If, however, there is some movement and he feels pressure with the fingers then you may be sure that the spinal cord in the neck is not seriously hurt. If the injury has taken place with regard to the vertebræ of the dorsal region then the same symptoms should be looked for in the feet and a similar judgment passed. If when he lies on his back he is incapable of retaining the contents of his intestines and gas passes from him involuntarily and if he is incapable of urinating then beyond all doubt the case is mortal."

When luxations of the cervical vertebræ occur he suggests a very curious and rather elaborate technic for the reduction of the dislocation and the reposition of the vertebræ in their proper relations. He adds very naively, "that is, of course, if death does not supervene." The mouth of the patient should be opened so that a piece of wood may be held between the teeth and a bandage wrapped around this and beneath the jaw as well as at the back of the head. The physician should then place one foot on one shoulder of the patient, the other on the other and should pull the head straight upwards until reduction takes place. In dislocations of the dorsal vertebræ the manipulations for reduction are the same as those which are to be found in Hippocrates. Most of the other dislocations as of the clavicle, the shoulder and elbow are treated in the same manner as has been suggested for them by the predecessors of many centuries before. These are the injuries of civil life in which the military experience of the moment had had no special significance.

## CANCER AND LUPUS

Theodoric had evidently had considerable experience with cancer and describes the various forms and the difficulty of treating them. They occur particularly in the breasts of women in connection with the nipples, and he seems to have felt that the conditions of use of these organs had something to do with the occurrence of cancer in them. Apparently the next most frequent place of the occurrence of cancer in women, in his experience, was in the uterus, though he speaks of this form of cancer as *occultus in matrice*, that is latent or hidden in the uterus, so that manifestly examinations were not made and yet the condition that developed was recognized as due to cancer. He speaks also of round ulcers with hollow edges and hardened raised lips (labia) which are very difficult to heal. He describes these as occurring particularly on the legs and had evidently seen leg ulcers develop malignant tendencies. He constantly talks of melancholy humors associated with these cancers and had beyond all doubt noted what we call cancer cachexia.

He describes two kinds of lupus and was evidently quite familiar with that affection. One of these is chronic and involves particularly the nose and mouth, often growing slowly, beginning as a pustule and remaining small for a whole year or more. He discusses why this was called *noli me tangere* by the old surgeons, that is, "don't touch me," and says it was either because there was distinct contagion associated with it, or else that if touched or irritated the humors flowed more to the part and so the disease was increased. The only treatment that he has found of any service, and it is very evident that some of the slow running cancers of the face are grouped in under the term lupus, were various caustics. The other form of lupus of which he speaks is acute, causes profound corrosion and often runs a very rapid course and must probably be considered, as Gurlt thinks, to have been some form of hospital gangrene. Cauterization was sometimes effective but a *medicamen acutum*, that is, some rapidly acting severe mode of treatment, was the only one presenting any hope of real relief from the condition.

## THROAT AND NOSE SURGERY

Tonsillitis is a subject on which Theodoric dwells rather particularly. He calls them branchi or branci, which recalls our word for

branchial arches, and says that occasionally they swell, producing the appearance of two almonds as it were. For this tonsillitis gargles should be used and if the patient is not relieved then recourse should be had to surgery. For this he suggests that the patient should be placed sitting before the surgeon with open mouth, his tongue being depressed with an instrument so that the surgeon may be able to see the tonsils well and having grasped the one to be operated on with an iron or bronze hook incise it with a knife. The pillars of the fauces which stand beside the tonsils should be left uninjured. This was evidently a description of an opening of a tonsillar abscess. Theodoric believed, however, in much more extensive operation on the tonsils if that were necessary. He says if the inflammation should go on to a severe degree (*putredo*) the incision in the tonsil should be so made that with instruments fashioned for this purpose they should be forced outward and entirely plucked out by a radical operation. In some of these cases, however, tonsils should be cauterized with a hot iron or gold instrument.

In elongation of the uvula where symptoms of irritation of the region occurs powder should be used or gargles employed to shrivel the organ somewhat. "If, however, it cannot be shriveled it should be caught in forceps made for this purpose and snipped off, care being taken, however, not to touch the roots of the uvula." It is not always clear just what Theodoric's expression means, for he describes a condition involving some carunculae in the neighborhood of the epiglottis which may obstruct the "tracheal artery" and hinder the voice. When the voice is thus suppressed only surgery can be of assistance. Gurlt seems inclined to think that in this description Theodoric referred to œdema of the glottis and the danger of it and the necessity for surgical intervention.

Theodoric has a good deal about angina, a term under which was included apparently abscesses of various kinds in the region of the pharynx, œsophagus and larynx, as well as evidently retropharyngeal and retroœsophageal abscesses. In the worst form of angina, pus forms in the space between the trachea and the œsophagus, which is called the isthmus, and this affection is extremely fatal and its cure must be left to God alone. In the second form, retropharyngeal abscess, part of the material is ejected and while some of it remains it is possible to

see the tumor which it forms and to treat it, and this kind of angina is less malignant. He suggests that when such an abscess produces disturbance by allowing infectious material, *saries*, to get into the body it is useful with the finger or with some instrument to rupture the abscess and cause it to be ejected. He said that in some of these cases he had cured patients in this way. He even suggests that a rather good sized piece of salt beef of the size of a large chestnut and only half cooked should be fastened to a piece of silk and swallowed, the surgeon retaining the silk thread and pulling out the swallowed piece suddenly and with violence with the idea of bringing about a rupture of the abscess.

With regard to goitre evidently Theodoric had had a good deal of experience and he suggests a number of means by which the goitre might be reduced. Most of these are such remedies as would be expected to have an effect on the mind rather than on the tissues. Even in our day there are traditions among country people that goitrous swellings may be reduced by wrapping a snake's skin around the neck or still more by wrapping a dead snake itself around over the goitre, or by the touch of the hangman's rope, or by a number of remedies that have no physical influence. As a matter of fact cases of exophthalmic goitre are often influenced very favorably by psychotherapy and it is evidently with these that Theodoric had had some successful experiences.

He says, however, that if by such means the patient cannot be cured recourse must be had to surgery. He suggests puncture with a hot iron or the making of a seton, and if these means do not help then radical surgery should be attempted, provided, however, the goitre is not too rich in blood-vessels. The skin above it should be cautiously cut longitudinally and the goitre itself lifted out with a hook and with the finger be brought out of the wound. He warns about the danger of serious hemorrhage, however, and adds that unless the goitre is completely removed even from a small particle that remains there may be regrowth or the patient may suffer as much as before.

The first chapter of the third book treats of fistulas in general. The mediæval surgeons studied fistulæ rather carefully. There is a famous English surgeon of the early thirteenth century, John of Ardern, who has a whole book devoted to this subject and who is

looked upon as a specialist in it. Among the special fistulas Bishop Theodoric treats of fistula of the lachrymal duct as well as mandibular fistula apparently connected either with abscess of the teeth or with caries of the jaw bone. He also had a special chapter devoted to fistulas of the neck, some of them superficial and evidently connected with tuberculous glands but others much deeper and more serious. He evidently refers to pathological conditions and neoplastic complications developing from the gill slits or primitive branchial arches. Theodoric warns that some of these are situated so deeply that they may have intimate surgical relations with the deep-lying arteries of the neck and that, therefore, their operative treatment must only be undertaken with the greatest foresight and care.

#### ABDOMINAL SURGERY

Suture of the abdominal wall is interesting in Theodoric's discussion of it because he suggests that the sutures can be carried through all the tissues from without inwards and then from within outwards on the other side, the parts being brought tightly together, or that separate portions of the abdominal wall may be brought together separately.

As regards wounds of the intestines four points of special care were to be noted. First, the contamination of the wound and its neighborhood with fecal masses was to be guarded against. Secondly, the wound was to be thoroughly cleansed. Thirdly, the intestines must be put back and, fourthly, special care must be taken of the after-treatment by position in bed and the like. In order to sew the intestine together a metal cylinder of such a size that it would fit the intestines properly should be introduced. The intestines should be sewed together above this with a very fine needle and with extremely fine thread made of the intestines of animals (our cat-gut). If this cannot be obtained, however, a fine silk thread should be used which is first washed well in warm wine, the intestines also being washed in this. After the suturing the intestines themselves should be very carefully cleansed with a soft linen plegget used just as delicately as possible. When the intestines have been cleansed and dried they should be put back, as was said above, and the wound should be sewed up.

Theodoric was very explicit in his declaration that wounds or incisions of the abdomen should not be left open for drainage, or observation purposes, or anything else, with the idea that the closure of the intestines should be waited for. A number of his predecessors and particularly Bruno of Longoburgo had recommended this, but Theodoric insisted that there were many dangers connected with the practice. First, as often as the patient's bandages were changed the exterior air may find an entrance and this will cause torments and pains in the intestines. Suffering will be inflicted and recovery will be delayed and even because of the conditions produced serious consequences may result. Secondly, when the wound is allowed to remain open the intestine may find its way out and this may have unfortunate effects. The wound should always be sewed up then and the patient should be given a clyster of warm wine and should be so placed in bed that the wound in the abdomen is always the highest part of the body.

The word used by Theodoric for the hollow tube to be placed inside of the intestines before sewing them up is called in Latin *sambucus*. This would be the musical instrument known as the sackbut or psaltery in Scriptural language, and evidently some portion of it made a suitable inner tube for this purpose. Apparently this tube is left within the intestines when they are sewed up. This is not surprising, at least not so surprising as it might possibly seem for those who think of any such instrumental aids, as of much more recent invention, once it is realized that a little later several different kinds of tubes for intestinal surgical work are described in the surgical text-books. Hugo von Pfolspeundt whose book on Military Surgery, *Field Ertznee*, was one of the first surgical books ever printed, being indeed among the earliest incunabula of printing (about 1460), described a silver tube with two flanges which he used in his intestinal surgical work. He said that it should be permitted to remain in the intestines and would eventually be passed out. He declared that he had often seen patients' lives saved by its use. Before him the Brancas in Italy, great surgeons, father and son, who are famous for their plastic work in the remaking of noses and lips, suggested that when wounds of the intestines occur if the intestine was not completely cut across it should be and then the trachea of an animal, freshly removed,

should be inserted in the intestine and the severed ends of the gut being brought together above this tube should be carefully coaptated and sutured. They declared that the trachea would remain *in situ* and retain its form long enough to enable the intestines to agglutinate and yet neither prevent intestinal peristalsis nor the flow of material through the intestines and then eventually be completely absorbed so that there was no necessity of waiting for it to pass out—a passage which was often delayed in the case of metal instruments and sometimes complicated the after course of these cases very much.

#### GENITO-URINARY AND DERMATOLOGIC SURGERY

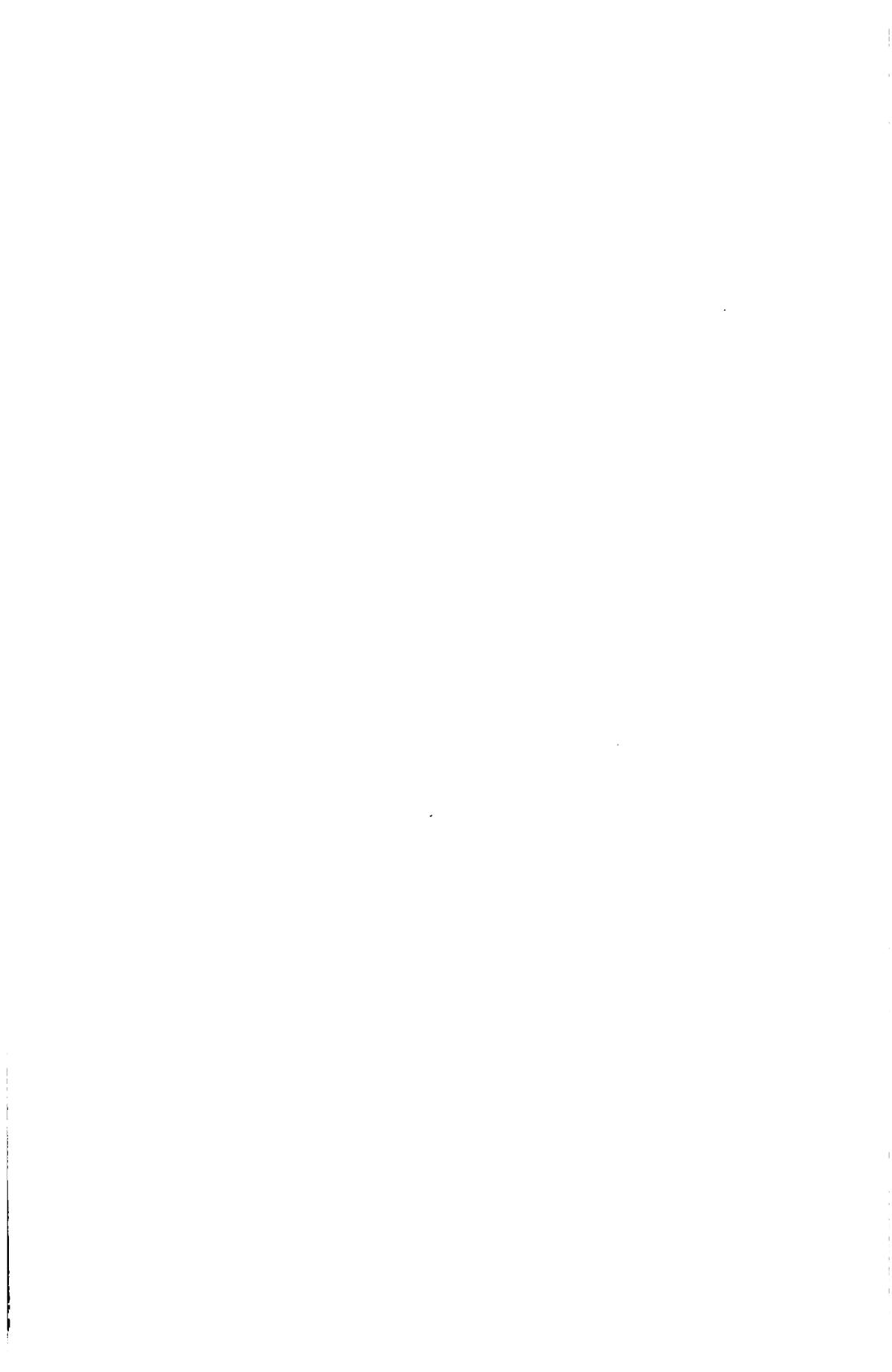
Theodoric has the technic for the treatment of dropsy, of hernia, of scrotal injuries, of hydrocele, sarocele, orchitis, relaxation of the scrotal tissues and other genito-urinary special affections. He has nearly a whole chapter on anal fistula and on various conditions that occur in connection with the anus. He has a chapter on renal and vesical calculi and while he was confident that certain remedies would cause the stone to dissolve he also knew that surgical procedures and especially lithotomy was sometimes recorded.

He has chapters on scabies and pruritus, on pimples on the face, on impetigo and serpigo, on morphea and various discolorations of the body, on facial tumors, on overgrowth of the hairs in the nose and loss of hair in the eyebrows, as well as warts, pannaritiae, leprosy, elephantiasis, alopecia, leontiasis, and has various therapeutic suggestions for these affections.

Theodoric's treatment of ganglion (cyst of a tendon sheath) was by a sudden blow given as follows: "If there is a node in the back of the hand probably the best surgical custom in the matter is to take the hand of the patient and with the other hand strike upon the node with a small plate or some similar hard object, repeating the percussion a second and third time if it seems best until the follicle ruptures and the place of the node remains vacant." After that a plaster made of styptic materials should be applied. Theodoric and his father had had some experiences with large lipomata which were of such a size that they feared to remove them and yet found that their removal was accomplished rather easily and without any serious consequences. Small tumors of the eyelids and nodes of the head,

evidently wens and the like were to be removed whenever they were the cause of much annoyance to the patient. He says, however, that his custom was never to advise operations for these tumors, large or small, of the head, unless there was very good reason, whenever there could be any danger in the operation. He was evidently, in spite of the linear scars of which he boasted and the uniformly excellent results without laudable pus, a very conservative surgeon.

Theodoric had many distinguished successors in the thirteenth century who accomplished much for the development of their specialty. Perhaps the best known of these was William of Salicet, also called the Master of Piacenza, because he was born there. William was a pupil of Buono di Garbo and the teacher of Lanfranc, the Italian surgeon, who did such magnificent work at the University of Paris in the second half of the thirteenth century. William of Salicet seems to have had no little of his experience as a military surgeon, as was true also of Lanfranc, and this group of men represent the most distinguished among the great university teachers of surgery in the thirteenth century. They were followed in the fourteenth by Guy de Chauliac, who was the Papal physician of the Popes while they were at Avignon, and Guy, like Theodoric, was an ecclesiastic and held the office of Canon toward the end of his life. The whole story of the surgery of this period is interesting mainly because it is such a contradiction of what we have been accustomed to think of as the history of surgery at the end of the Middle Ages.



# INDEX TO VOLUME III

## (TWENTY-EIGHTH SERIES)

### A

Abdomen, upper, malignant disease of, 117  
Abdominal surgery, history of, 290  
Abortion, 100  
    curettage after, 102  
    endometritis after, 100  
    incomplete, 108  
Acromegaly, acute, 154  
Alkali to neutralise gastric secretion, 52  
Anæsthesia at battle front, 210  
    consideration of its use at the front, 226  
    ether, influence on pain, 280  
    immediate use of, in war, 212  
    in Europe, 210  
    local, influence on pain transmission, 281  
    methods of obtaining, 216  
    schools for instructing in, 218  
    speedy induction of, in war, 214  
    spinal, 224  
    training in the giving of, 217  
    vomiting after, 212  
Anaphylaxis from eggs, 95  
Aneurysm, thoracic, 80  
Aneurysmal hematoma, 55  
    clinical aspects of, 59  
    diagnosis of, 59  
    infection of, 62  
    treatment of, 63  
    types of, 55  
Appendicitis, 133  
    acute, 119  
    and peritonitis, 141  
    chronic, 118  
Arteriosclerosis, cerebral, 175  
Atrophy of optic nerve, 186  
Auricular fibrillation, 20  
    causes of, 28  
    digitalis in, 36  
    duration of, 30  
    effect on heart, 30  
    nature of, 28  
    recognition of, 33  
    signs of, 26  
    symptoms of, 26  
    treatment of, 35  
Auscultation of stomach, 49

### B

Bacteriology of infectious endocarditis, 17  
Birth-palsy, 144

Brain, history of surgery of, 284

Bronchiectasis, 72

Bronchitis, acute, 70  
    chronic, 71

### C

Cæcum, carcinoma of, 135  
    mobile, 114  
Carcinoma, gastric, 260, 263  
    of cæcum, 135  
    of lungs, 75  
Cardiac contraction, mechanism of, 20  
Cerebral arteriosclerosis, 175  
Cervical lymph nodes, removal of, 118, 125  
Children, skin reactions in, 82  
Chloroform, 220  
    ether mixtures, 215, 219  
Cholecystic disease, adhesions in, 266  
Cholecystitis, 266  
    chronic, 187  
Cholelithiasis and pancreatitis, chronic, 260  
Choleliths, 266  
Cholemia, familial, 158  
    blood in, 165  
    blood serum in, 169  
    common form of, 165  
    diagnosis of, 168  
    etiology of, 159  
    gastro-intestinal form of, 165  
    hemorrhagic form of, 165  
    hemorrhagic manifestations of, 165  
    liver in, 165  
    nervous form of, 165  
    pathologic physiology of, 168  
    prognosis in, 168  
    spleen in, 165  
    treatment of, 170  
    types of, 165  
    urine in, 164  
Christian, Henry A., 1  
Complement fixation test in tuberculosis, 70  
Conduction system of heart, 22  
Cotyloid cavity, fracture of, 248  
Cumston, Charles Greene, 55  
Curettage after abortion, 102  
Cutaneous test for food poisons, 96

### D

Dermatologic surgery, history of, 292  
Diet, history of, 283

Digitalis in auricular fibrillation, 36  
 Diphtheria, immunity to, as shown by Schick test, 86  
 Distomatosis, pulmonary, 79  
 Duodenal adhesions, 266

**E**

Eggs, anaphylaxis from, 95  
 Endocarditis, acute vegetative, 1  
     chronic infections, 16  
     diagnosis of, 10  
     infectious, bacteriology, 17  
 Endometritis after abortion, 100  
 Epilepsy, senile, arteriosclerosis and, 175  
     causes of, 175  
     hemorrhage and, 176  
     intoxication and, 177, 181  
     meninges in, 176  
     thyroid inefficiency in, 184  
 Erdmann, John F., 113  
 Ether, 219  
     anesthesia, effect of, on pain, 230  
 Ethyl chloride, 215, 228

**F**

Familial cholemia, 158  
     pathogenic study of, 158  
 Femur, intrapelvic dislocation of head of, 248  
 Fibroid uterus, 126  
 Fisher, Lewis, 200  
 Flagg, P. J., 210  
 Food reactions, 95  
     digestion, determination of end of, 46  
 Forceps operation, median, 107  
 Foster, G. S., 229  
 Fractional gastric tube, 40  
     introduction of, 40  
     length of, 48  
     medication through, 47  
     Murphy drip through, 48  
     principles of, 40  
 Fracture of cotyloid cavity, 248  
     etiology of, 250  
     signs of, 253  
     symptoms of, 253  
     treatment of, 258  
 Fractures, compound, history of, 288

**G**

Gall stones, 266  
     intrahepatic, 181  
 Gastric analysis, fractional, 45  
     carcinoma, 260, 263  
     secretion, neutralization by alkali, 52  
     therapy, direct, 47  
     tube, fractional, 40  
 Gastro-duodenal tube, double, 52, 53  
 Genito-urinary surgery, history of, 292  
 Goitre, 289

**H**

Headache, sinusitis and, 200  
 Heart beat, stimulus production of, 20  
     conduction system of, 22  
     effect of auricular fibrillation on, 80  
 Hemorrhage, history of treatment of, 282  
 Herniotomy, 122  
 Hess, Julius H., 82  
 History of abdominal surgery, 29  
     brain surgery, 284  
     dermatologic surgery, 292  
     genito-urinary surgery, 292  
     military surgery, 274  
     nose surgery, 287  
     spinal surgery, 280  
     throat surgery, 287  
     treatment of fractures, compound, 283  
     hemorrhage, 282  
     wounds, 282

Hydrochloric acid given through fractional tube, 51

Hymen, imperforate, 180  
 Hyperacidity, Sippy treatment of, 52  
 Hypnosis, 225

**I**

Intrahepatic stones, 181

**L**

Laboratory aids in tuberculosis, 68  
 Landis, H. R. M., 66  
 Laparotomy, exploratory, 115, 183  
 Lavage of stomach, 46  
 Levinson A., 82  
 Loewenberg, S. A., 154  
 Luetin test, 89  
     vs. Wassermann test, 93  
     reactions, 90  
     papular form, 90  
     pustular form, 90  
     torpid form, 90

Lungs, malignant disease of, 75

Lydston, G. Frank, 148

Lymph nodes, cervical, 118, 125

**M**

MacKechnie, Hugh N., 187  
 Malignant disease of lungs, 75

Margarine, E., 186

Marquez, Maurice, 248

Matorine, O., 174

McPherson, Ross, 100

Meninges, thickening of, 176

Military anesthesia, 210  
     hospital zones, 210  
     surgery, 274

Mitral stenosis, 23, 80

Morphine, influence of, on pain, 200

Murphy drip with the fractional gastric tube, 48

Mycotic infection of lungs, 78

**N**

Narcotics, influence on pain by, 280  
 Nerve blocking in surgery, 229  
     optic, post-traumatic atrophy of, 186  
 Nervous system, effect of pain on, 229  
 Neuritis, optic atrophy from, 196  
 Nitrous oxide gas, 212, 221  
     oxygen, 221  
 Nose, history of surgery of, 287  
 Novocaine, 281

**O**

Obstetrical forceps, application of, 109  
 Operating room, gray dressings for, 104  
 Ophthalmology, 186  
 Optic atrophy from neuritis, 196  
     papillary stasis, 196  
     nerve, contusion of, 196  
     post-traumatic atrophy of, 186  
     types of post-traumatic atrophy of, 195

**P**

Pain, conclusions on the effects of anesthetics on, 233  
     control of, by anesthetics, 234  
     effects of, on the nervous system, 229  
     influence of ether on, 238  
         local anesthesia on, 281  
         narcotics on, 280  
         nerve transmission, 288, 289, 241, 242

Palsy, birth, 144

Pancreatitis, chronic, 260, 269

Paralysis, birth, 144

Pathology of pulmonary tuberculosis, 68

Patterson, Ross V., 20

Perineorrhaphy, 105

Peritonitis and appendicitis, 141

Pneumoconiosis, 74

Pneumonia, lobar, 76

Polioencephalitis superior, 248  
     operative treatment of, 248

Pope, Curran, 260

Prostate, enlarged, non-surgical treatment of, 148

Pyloric adhesions, 266

**R**

Rehfuss, Martin H., 40

Rey, Paul, 158

Reynolds, Cecil E., 248

Rhinology, 200

**S**

Sarcoma of lungs, 75  
 Schick test, 82  
     evidence of immunity from, 86  
 Scopolamine, influence of, on pain, 230  
 Shock, 229  
 Sigmoid, malignant disease of, 115  
 Sinusitis and headache, 200  
 Sinus infection, treatment of, 204  
 Sippy treatment of hyperacidity, 52  
 Skin reactions in children, 82

Skull, history of surgery of, 284

Spinal anæsthesia, 224  
     surgery, history of, 286

Stomach, auscultation over, by means of fractional tube, 49

carcinoma of, 260, 263

determination of velocity of secretion, 50

empty, 44

interdigestive phase, 44

lavage of, 46

rest phase, 44

syphilis of, 265

syphon method of examining, 50

tube, fractional, 40

Stovaine, 224

Surgery, 210

abdominal history of, 290

dermatologic history of, 292

history of genito-urinary, 292

military, 274

of nose and throat, history of, 287

skull and brain, history of, 284

spinal, history of, 286

Syphilis of stomach, 265

pulmonary, 77

**T**

Theodosic of Lucca, 274

Thoracic aneurysm, 80

Throat, history of surgery of, 287

Thyroid inefficiency and senile epilepsy, 184

Thyroidectomy, 128

Tuberculin, 69

Tuberculosis, pulmonary, 66

aneurysm and, 80

bronchiectasis and, 72

bronchitis and, 70

complement fixation test in, 70

conditions simulating, 66

distomatosis and, 79

history in, 66

laboratory aids in, 68

malignant disease and, 75

mitral stenosis and, 80

myotic infections and, 78

pathology of, 68

pneumoconiosis and, 74

pneumonia and, 76

syphilis and, 77

tuberculin and, 69

**U**

Uterus, fibroid, 176

**V**

Varicose veins, excision of, 123

**W**

Walsh, James J., 274

Wassermann test *vs.* luetin test, 98

Willmorth, A. D., 260

Wounds, history of treatment of, 282











2 GAL 550

COUNTWAY LIBRARY



HC 2EMJ 0

